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=> file medline, uspatful, dgene, embase, wpids, biotechds, biosis, scisearch

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=> s TLR-8
L1          82 TLR-8

=> s l1 and agonist
L2          37 L1 AND AGONIST

=> s l1 and (detection method)
   5 FILES SEARCHED...
L3           0 L1 AND (DETECTION METHOD)

=> s l2 and (test compound)
L4           9 L2 AND (TEST COMPOUND)

=> d l4 ti abs ibib tot

L4   ANSWER 1 OF 9  USPATFULL on STN
TI     Methods and products based on oligomerization of stress proteins
AB     In one aspect, the invention provides methods for determining the
       biological activity of heat shock proteins or heat shock protein-peptide
       complexes based on the ATPase activity or the multimeric structure of
       the heat shock proteins or heal shock protein-peptide complexes, and
       methods for screening agents that modulate the biological activity of
       heat shock proteins or heat shock protein-peptide complexes. In another
       aspect, the invention provides complexes, compositions and methods for
       enhancing the immunogenicity of a heat shock protein or a complex
       comprising a heal shock protein and an antigenic molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:       2005:254901  USPATFULL
TITLE:                  Methods and products based on oligomerization of stress
                        proteins
INVENTOR(S):            Zabrecky, James R., Waltham, MA, UNITED STATES
                        Liu, Chuanling, Haverhill, MA, UNITED STATES
                        Monks, Stephen A., Arlington, MA, UNITED STATES
                        Wasserman, Andrew, North Andover, MA, UNITED STATES
                        Srivastava, Pramod K., Avon, CT, UNITED STATES

                             NUMBER       KIND    DATE
                        --------------- ------ --------
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PATENT INFORMATION:	US 2005221395	A1	20051006	
APPLICATION INFO.:	US 2003-506097	A1	20030228	(10)
	WO 2003-US6298		20030228	
			20050314	PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-60361257	20020228

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017, US
NUMBER OF CLAIMS: 81
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 5793
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 2 OF 9 USPATFULL on STN
TI Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
AB The invention pertains to murine TLR9 and related TLR9s which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR9. The invention further relates to methods of using such murine and non-murine TLR9 nucleic acids and polypeptides, especially in methods for screening for agonists and antagonists of immunostimulatory CpG nucleic acids. Also included are murine TLR9 inhibitors which inhibit murine TLR9 activity by inhibiting the expression or function of murine TLR9. In a further aspect the present invention pertains to murine TLR7 and murine TLR8, as well as related TLR7 and TLR8 molecules which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR7 and TLR8. Methods are included for screening for ligands of TLR7 and TLR8, as well as for inhibitors and agonists and antagonists of signaling mediated by TLR7 and TLR8.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:208942 USPATFULL
TITLE: Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
INVENTOR(S): Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
Lipford, Grayson, Watertown, MA, UNITED STATES
Wagner, Hermann, Eching, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005181422	A1	20050818	
APPLICATION INFO.:	US 2005-84777	A1	20050318	(11)
RELATED APPLN. INFO.:	Division of Ser. No. US 2001-954987, filed on 17 Sep 2001, PENDING			

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-233035P	20000915 (60)
	US 2001-263657P	20010123 (60)
	US 2001-291726P	20010517 (60)
	US 2001-300210P	20010622 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600

ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 9366
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 3 OF 9 USPATFULL on STN
TI Use of lectins to promote oligomerization of glycoproteins and antigenic molecules
AB The present invention relates to using lectin or lectin-like molecules to promote oligomerization of a glycoprotein or an immunologically and/or biologically active complex comprising glycoproteins. In particular, the invention provides compositions of a molecular complex comprising lectin molecules and immunologically and/or biologically active molecules. Methods of making such molecular complexes and methods of use of the compositions comprising such molecular complexes for the prevention and treatment of diseases, particularly cancer and infectious diseases, and for eliciting an immune response in a subject, are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:326886 USPATFULL
TITLE: Use of lectins to promote oligomerization of glycoproteins and antigenic molecules
INVENTOR(S): Zabrecky, James R., Waltham, MA, UNITED STATES
Monks, Stephen A., Arlington, MA, UNITED STATES
PATENT ASSIGNEE(S): Antigenics Inc. (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004258705	A1	20041223	
APPLICATION INFO.:	US 2004-789220	A1	20040227	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-450721P	20030228	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017
NUMBER OF CLAIMS: 70
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 5764
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 4 OF 9 USPATFULL on STN
TI Methods for using compositions comprising heat shock proteins or alpha-2-macroglobulin in the treatment of cancer and infectious disease
AB The present invention relates to methods and compositions for the prevention and treatment of infectious diseases, and cancers. The methods of the invention comprises administering (a) a composition comprising a population of complexes of antigenic proteins or antigenic peptides derived from antigenic cells or viral particles and one or more different heat shock proteins; and (b) a non-heat shock protein and non-alpha-2-macroglobulin-based treatment modality. The population or the protein preparation used to produce the antigenic peptides comprises at least 50% of the different proteins or at least 50 different proteins of the antigenic cells or viral particles. Methods for making antigenic peptides comprise digesting a protein preparation of antigenic cells, a cellular fraction thereof, or of viral particles with one or more proteases, or exposing the protein preparation to ATP, guanidium hydrochloride, and/or acidic conditions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:320575 USPATFULL
TITLE:	Methods for using compositions comprising heat shock proteins or alpha-2-macroglobulin in the treatment of cancer and infectious disease
INVENTOR(S):	Srivastava, Pramod K., Avon, CT, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004253228	A1	20041216	
APPLICATION INFO.:	US 2004-784012	A1	20040220	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-449001P	20030220 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017
NUMBER OF CLAIMS:	39
EXEMPLARY CLAIM:	1
LINE COUNT:	4653

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 5 OF 9 USPATFULL on STN

TI Methods and products for enhancing immune responses using imidazoquinoline compounds

AB The invention involves administration of an imidazoquinoline agent in combination with another therapeutic agent. The combination of drugs may be administered in synergistic amounts or in various dosages or at various time schedules. The invention also relates to kits and compositions concerning the combination of drugs. The combinations can be used to enhance ADCC, stimulate immune responses and/or patient and treat certain disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:201378 USPATFULL
TITLE:	Methods and products for enhancing immune responses using imidazoquinoline compounds
INVENTOR(S):	Krieg, Arthur M., Wellesley, MA, UNITED STATES Schetter, Christian, Hilden, GERMANY, FEDERAL REPUBLIC OF Bratzler, Robert L., Concord, MA, UNITED STATES Vollmer, Jorg, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF Jurk, Marion, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):	University of Iowa Research Foundation, Iowa City, IA, 52242 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003139364	A1	20030724	
APPLICATION INFO.:	US 2002-272502	A1	20021015	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-329208P	20011012 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS:	87
EXEMPLARY CLAIM:	1

NUMBER OF DRAWINGS: 25 Drawing Page(s)
LINE COUNT: 7027
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 6 OF 9 USPATFULL on STN
TI Methods of maturing plasmacytoid dendritic cells using immune response modifier molecules
AB The present invention relates to methods of maturing plasmacytoid dendrites cells using immune response modifier molecules. The present invention also relates to methods of detecting biological activities of matured plasmacytoid dendritic cells and methods of using mature plasmacytoid dendritic cells for therapeutic or prophylactic purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:194103 USPATFULL
TITLE: Methods of maturing plasmacytoid dendritic cells using immune response modifier molecules
INVENTOR(S): Tomai, Mark A., Woodbury, MN, UNITED STATES
Vasilakos, John P., Woodbury, MN, UNITED STATES
Stolpa, John C., St. Paul, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003133913	A1	20030717	
APPLICATION INFO.:	US 2002-229829	A1	20020828	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-316144P	20010830	(60)
	US 2002-370177P	20020405	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 93
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 2566
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 7 OF 9 USPATFULL on STN
TI Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
AB The invention pertains to murine TLR9 and related TLR9s which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR9. The invention further relates to methods of using such murine and non-murine TLR9 nucleic acids and polypeptides, especially in methods for screening for agonists and antagonists of immunostimulatory CpG nucleic acids. Also included are murine TLR9 inhibitors which inhibit murine TLR9 activity by inhibiting the expression or function of murine TLR9. In a further aspect the present invention pertains to murine TLR7 and murine TLR8, as well as related TLR7 and TLR8 molecules which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR7 and TLR8. Methods are included for screening for ligands of TLR7 and TLR8, as well as for inhibitors and agonists and antagonists of signaling mediated by TLR7 and TLR8.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:152857 USPATFULL

TITLE: Process for high throughput screening of CpG-based immuno-**agonist**/antagonist

INVENTOR(S): Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
Lipford, Grayson, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF
Wagner, Hermann, Eching, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003104523	A1	20030605	
	US 6943240	B2	20050913	
APPLICATION INFO.:	US 2001-954987	A1	20010917	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-233035P	20000915 (60)
	US 2001-263657P	20010123 (60)
	US 2001-291726P	20010517 (60)
	US 2001-300210P	20010622 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS: 120
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 6814
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 8 OF 9 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject.
AN 2003-393260 [37] WPIDS
AB WO2003020889 A UPAB: 20030612
NOVELTY - Obtaining (M1) a population of mature dendritic cells, comprises administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a cell population (I) obtained by (M1);

(2) enhancing (M2) antigen presentation by dendritic cells in vitro, comprising:

(a) exposing an isolated dendritic cell population to an antigen;

(b) contacting the isolated dendritic cell with IRM; and

(c) allowing the dendritic cell to process and present the antigen;

(3) an isolated dendritic cell population (II) produced by (M2);

(4) detecting (M3) cytokine production, expression of co-stimulatory markers, or expression of chemokine receptors by a plasmacytoid dendritic cell (pDC), comprising:

(a) contacting isolated pDC with IRM for inducing the plasmacytoid dendritic cell to produce one or more cytokines selected from interleukin (IL)-8, IP-10, IL-6, macrophage Inflammatory Protein 1 alpha (MIP-1 alpha), and interferon (IFN)- omega , or to express one or more co-stimulatory marker or chemokine receptor; and

(b) detecting production of one of the cytokines, co-stimulatory marker, or chemokine receptor by the dendritic cell;

(5) enhancing (M4) survival of isolated plasmacytoid dendritic cells, comprising:

(a) contacting a population of isolated pDCs with an IRM in an amount effective for enhancing survival of the pDCs; and

(b) incubating pDCs under conditions so that 30 % of pDC survive for 48 hours;

(6) identifying (M5) a compound that selectively induces production of a chemokine receptor by pDCs, comprising:

(a) obtaining a population of cells that includes both inflammatory cytokine producing cells and pDCs;

(b) contacting the population of cells with a **test compound**;

(c) determining the amount of chemokine receptor present in the population of cells contacted with the **test compound**;

(d) determining the amount of inflammatory cytokine(s) present in the population of cells contacted with the **test compound**; and

(e) identifying the **test compound** as a selective inducer of the chemokine receptor if the chemokine receptor is present in the population of cells after contact with the **test compound** in an amount 3 times greater than the amount of inflammatory cytokine(s) present in the population of cells;

(7) preparing (M6) a cell population enriched for cells that express a chemokine receptor, comprising:

(a) contacting pDC with IRM for inducing pDC to express one or more chemokine receptor; and

(b) enriching the cell population for cells that express a chemokine receptor;

(8) a population of pDCs enriched for cells that express chemokine receptors prepared by (M6); and

(9) a cellular adjuvant (III) prepared by maturing pDCs in vitro by treating dendritic cells with IRM, and exposing mature pDCs to antigens associated with the disease.

ACTIVITY - Neuroprotective; Antidiabetic; Antirheumatic; Antiarthritic; Antiinflammatory; Antipsoriatic; Tuberculostatic; Antileprotic; Protozoacide; Fungicide; Virucide; Antiparasitic; Antibacterial; Dermatological; Antiallergic; Anti-HIV; Antiasthmatic; Immunosuppressive; Nootropic; Cardiant; Cytostatic; Muscular; Immunostimulant.

MECHANISM OF ACTION - Ex vivo gene therapy; Vaccine. No biological data is given.

USE - (M1) And another new method (M2) are useful for treating a disease, by:

(a) contacting an isolated pDC with IRM for inducing pDC to express chemokine receptors;

(b) contacting the population of pDC with an antigen associated with the disease;

(c) enriching the cell population for cells expressing a high level of a chemokine receptor; and

(d) administering the enriched cell population to a patient.

A cell population (I) obtained by (M1), or a cellular adjuvant (III) is useful for treating a disease. (M1) Is useful for preparing a cellular adjuvant for the treatment of a disease, by maturing pDCs in vitro, and exposing mature pDCs to antigens associated with the disease. The disease is a neoplastic disease and the antigen is derived from neoplastic cells. The disease is caused by an infectious agent and the antigen is derived from the infectious agent. The disease is a Th2-mediated disease (claimed). (I) Is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., acquired immunodeficiency syndrome (AIDS), in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. (III) Is useful for provoking an anti-tumor immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and

heart disease. (I) Is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation.

Dwg.0/5

ACCESSION NUMBER: 2003-393260 [37] WPIDS
DOC. NO. CPI: C2003-104375
TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject.
DERWENT CLASS: B04 D16
INVENTOR(S): STOLPA, J C; TOMAI, M A; VASILAKOS, J P
PATENT ASSIGNEE(S): (MINN) 3M INNOVATIVE PROPERTIES CO
COUNTRY COUNT: 102
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003020889	A2	20030313	(200337)*	EN	84
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW					
US 2003133913	A1	20030717	(200348)		
EP 1427445	A2	20040616	(200439)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
AU 2002329892	A1	20030318	(200452)		
JP 2005501550	W	20050120	(200508)		143
IN 2004000453	P4	20041218	(200533)		
MX 2004001972	A1	20050301	(200568)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003020889	A2	WO 2002-US27393	20020828
US 2003133913	A1 Provisional	US 2001-316144P	20010830
	Provisional	US 2002-370177P	20020405
		US 2002-229829	20020828
EP 1427445	A2	EP 2002-766145	20020828
		WO 2002-US27393	20020828
AU 2002329892	A1	AU 2002-329892	20020828
JP 2005501550	W	WO 2002-US27393	20020828
		JP 2003-525593	20020828
IN 2004000453	P4	WO 2002-US27393	20020828
		IN 2004-CN453	20040301
MX 2004001972	A1	WO 2002-US27393	20020828
		MX 2004-1972	20040227

FILING DETAILS:

PATENT NO	KIND	PATENT NO

EP 1427445 A2 Based on WO 2003020889
AU 2002329892 A1 Based on WO 2003020889
JP 2005501550 W Based on WO 2003020889
MX 2004001972 A1 Based on WO 2003020889

PRIORITY APPLN. INFO: US 2002-370177P 20020405; US
2001-316144P 20010830; US
2002-229829 20020828

L4 ANSWER 9 OF 9 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject;
mature dendrite cell production and immune response modifier moelcule for use in disease gene therapy and vaccine
AN 2003-16040 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Obtaining (M1) a population of mature dendritic cells, comprises administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) a cell population (I) obtained by (M1); (2) enhancing (M2) antigen presentation by dendritic cells in vitro, comprising: (a) exposing an isolated dendritic cell population to an antigen; (b) contacting the isolated dendritic cell with IRM; and (c) allowing the dendritic cell to process and present the antigen; (3) an isolated dendritic cell population (II) produced by (M2); (4) detecting (M3) cytokine production, expression of co-stimulatory markers, or expression of chemokine receptors by a plasmacytoid dendritic cell (pDC), comprising: (a) contacting isolated pDC with IRM for inducing the plasmacytoid dendritic cell to produce one or more cytokines selected from interleukin (IL)-8, IP-10, IL-6, macrophage Inflammatory Protein 1alpha (MIP-1alpha), and interferon (IFN)-omega, or to express one or more co-stimulatory marker or chemokine receptor; and (b) detecting production of one of the cytokines, co-stimulatory marker, or chemokine receptor by the dendritic cell; (5) enhancing (M4) survival of isolated plasmacytoid dendritic cells, comprising: (a) contacting a population of isolated pDCs with an IRM in an amount effective for enhancing survival of the pDCs; and (b) incubating pDCs under conditions so that 30 % of pDC survive for 48 hours; (6) identifying (M5) a compound that selectively induces production of a chemokine receptor by pDCs, comprising: (a) obtaining a population of cells that includes both inflammatory cytokine producing cells and pDCs; (b) contacting the population of cells with a **test compound**; (c) determining the amount of chemokine receptor present in the population of cells contacted with the **test compound**; (d) determining the amount of inflammatory cytokine(s) present in the population of cells contacted with the **test compound**; and (e) identifying the **test compound** as a selective inducer of the chemokine receptor if the chemokine receptor is present in the population of cells after contact with the **test compound** in an amount 3 times greater than the amount of inflammatory cytokine(s) present in the population of cells; (7) preparing (M6) a cell population enriched for cells that express a chemokine receptor, comprising: (a) contacting pDC with IRM for inducing pDC to express one or more chemokine receptor; and (b) enriching the cell population for cells that express a chemokine receptor; (8) a population of pDCs enriched for cells that express chemokine receptors prepared by (M6); and (9) a cellular adjuvant (III) prepared by maturing pDCs in vitro by treating dendritic cells with IRM, and exposing mature pDCs to antigens associated with the disease.
BIOTECHNOLOGY - Preferred Method: Mature dendritic cells are

isolated from a blood sample of a subject. The amount of immune response modifier molecule administered to the subject is 0.001 mg/kg. The dendritic cells are pDCs. The antigen is derived from neoplastic cells, infectious agent, or is recombinantly derived. The immune response modifier molecule is an imidazoquinoline amine, imidazopyridine amine, 6,7-fused cycloalkylimidazopyridine amine, 1,2-bridged imidazoquinoline amine, thiazolo- and oxazolo-quinolinamine or pyridinamine, imidazonaphthyridine amine or tetrahydroimidazonaphthyridine amine, or their salts. The method further involves detecting the antigen presentation. The cytokines are IFN-gamma or IL-10. In (M3), the amount of IRM is provided at a concentration of 0.001 microM. Extracellular or intracellular cytokine, chemokine, and co-stimulatory marker are detected by flow cytometry or enzyme-linked immunosorbant assay. Cytokine, chemokine, and co-stimulatory marker production are detected by detecting mRNA that encodes the cytokine, chemokine, or co-stimulatory marker in the plasmocytoid dendritic cell. The co-stimulatory marker is cluster of differentiation (CD)80, CD86, CD40, or human leucocyte antigen (HLA)-DR. Expression of co-stimulatory marker is detected by detecting co-stimulatory marker on the cell surface of pDC. The chemokine receptor is CCR7. Detecting expression of a chemokine receptor, comprises detecting up-regulation of chemokine receptor expression or down-regulation of chemokine receptor expression. In (M4), 50 %, 70 % or 75 % of the plasmacytoid dendritic cells survive for 48 hours. In (M5), the amount of inflammatory cytokine(s) is determined from culture supernatants using an enzyme-linked immunosorbant assay or a bioassay. The amounts of chemokine receptor and inflammatory cytokine(s) are determined using Northern blotting, Western blotting, or real-time polymerase chain reaction (PCR). The inflammatory cytokine is tumor necrosis factor (TNF)-alpha or IL-12. The population of cells is contacted with the **test compound** at a concentration of 0.005 - 5 microM. (M6) Involves selectively removing cells that do not express chemokine receptor from the cell population, or: (a) contacting the cell population with a substrate that selectively binds cells that express a chemokine receptor to a substrate; (b) allowing the substrate to reversibly bind cells that express a chemokine receptor; (c) removing unbound cells; and (d) collecting the bound cells. The selective binding is adsorption or immunosorption.

ACTIVITY - Neuroprotective; Antidiabetic; Antirheumatic; Antiarthritic; Antiinflammatory; Antipsoriatic; Tuberculostatic; Antileprotic; Protozoacide; Fungicide; Virucide; Antiparasitic; Antibacterial; Dermatological; Antiallergic; Anti-HIV; Antiasthmatic; Immunosuppressive; Nootropic; Cardiant; Cytostatic; Muscular; Immunostimulant.

MECHANISM OF ACTION - Ex vivo gene therapy; Vaccine. No biological data is given.

USE - (M1) And another new method (M2) are useful for treating a disease, by: (a) contacting an isolated pDC with IRM for inducing pDC to express chemokine receptors; (b) contacting the population of pDC with an antigen associated with the disease; (c) enriching the cell population for cells expressing a high level of a chemokine receptor; and (d) administering the enriched cell population to a patient. A cell population (I) obtained by (M1), or a cellular adjuvant (III) is useful for treating a disease. (M1) Is useful for preparing a cellular adjuvant for the treatment of a disease, by maturing pDCs in vitro, and exposing mature pDCs to antigens associated with the disease. The disease is a neoplastic disease and the antigen is derived from neoplastic cells. The disease is caused by an infectious agent and the antigen is derived from the infectious agent. The disease is a Th2-mediated disease (claimed). (I) Is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., acquired immunodeficiency syndrome (AIDS), in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the

generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. (III) Is useful for provoking an anti-tumor immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) Is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells·e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation.

ADMINISTRATION - No administration details are given.

EXAMPLE - Human plasmacytoid dendritic cells (pDCs) were isolated from peripheral blood mononuclear cells (PBMC) by immunomagnetic bead positive selection. PBMC were incubated with pDC-specific antibodies, BDCA-2 or BDCA-4, and the labeled cells were collected. The positively selected cells were resuspended in X-Vivo 20 (RTM) medium. Human pDC were also enriched by negative selection from PBMC by depleting Lin+ cells. PBMC isolated from 120 ml whole blood were resuspended in 1 ml phosphate buffered saline (PBS), 1 % bovine serum albumin (BSA), 1 mM ethylenediaminetetraacetic acid (EDTA) and incubated with biotin-labeled antibodies specific for cluster of differentiation (CD)3, CD14, CD19, CD56 and in some case CD11b and CD11c, at a final concentration of 100 micrograms/ml for each antibody. After 15 minutes of incubation at 6 - 12 degrees Centigrade, the cells were washed and incubated with either streptavidin microbeads or anti-biotin microbeads for an additional 15 minutes at 6 - 12 degrees Centigrade. After washing, the unlabeled fraction was collected on Miltenyi (RTM) CS or LS columns and the cells were resuspended in X-Vivo 20 (RTM). The pDC population, HLA-DR+/CD123HI, was routinely 5 - 10 % of the final preparation as compared to 0.1 - 0.5 % of the staring PBMC population. Cells were incubated at 1 x 10 to the power of 6/ml in X-Vivo 20 (RTM) medium and stimulated with immune response modifiers (IRM) (4-amino-2-ethoxymethyl-alpha, alpha-dimethyl-1H-imidazo(4,5-c)quinoline-1-ethanol) for 1 hour. After stimulation, 1 microliter Brefeldin-A was added for every ml of cell culture medium. The cells were then incubated overnight at 37 degrees Centigrade with 5 % carbon dioxide, not exceeding 12 hours. The cells were washed and resuspended in Pharmingen (RTM) Stain Buffer-BSA two times. Fc receptors were blocked with ImmunoPure mouse immunoglobulin (Ig)G (100 ml/10 to the power of 6 cells in 100 microliters of staining buffer for 15 minutes at 4 degrees Centigrade). Cells were then washed with staining buffer and then stained for surface antigens (10 microliters antibody in 50 microliters staining buffer for 30 minutes at 4 degrees Centigrade). Cells were then washed and resuspended in cytofix/cytoperm to fix and permeabilized the cells. After washing with perm/wash solution, the cells were stained for intracellular cytokines with anti-tumor necrosis factor (TNF)-alpha or anti-interferon (IFN)-alpha fluorochrome-labeled antibodies for 30 - 45 minutes at 4 degrees Centigrade. Finally, the cells were washed and resuspended in staining buffer and analyzed using a FACSCAN FLOW (RTM) cytometer and CellsQuest (RTM) software. (84 pages)

ACCESSION NUMBER: 2003-16040 BIOTECHDS

TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject;
mature dendrite cell production and immune response modifier moelcule for use in disease gene therapy and vaccine

AUTHOR: TOMAI M A; VASILAKOS J P; STOLPA J C

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PATENT ASSIGNEE:  3M INNOVATIVE PROPERTIES CO
PATENT INFO:      WO 2003020889 13 Mar 2003
APPLICATION INFO: WO 2002-US27393 28 Aug 2002
PRIORITY INFO:    US 2002-370177 5 Apr 2002; US 2001-316144 30 Aug 2001
DOCUMENT TYPE:    Patent
LANGUAGE:         English
OTHER SOURCE:     WPI: 2003-393260 [37]


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E1            1     GORDEN WAGENER/AU
E2            1     GORDEN WILLIAM/AU
E3            0 --> GORDEN, K/AU
E4            1     GORDENCHUK K S/AU
E5            1     GORDENCHUK V D/AU
E6            6     GORDENCHUK V G/AU
E7            1     GORDENI D A/AU
E8            1     GORDENIN A/AU
E9            7     GORDENIN D/AU
E10         127     GORDENIN D A/AU
E11           1     GORDENIN D D/AU
E12           1     GORDENIN D L/AU

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E2            1     XIU ZY/AU
E3            0 --> XIU, X/AU
E4            1     XIUBIN H/AU
E5            4     XIUBIN HE/AU
E6            4     XIUBO L/AU
E7            1     XIUBO Y/AU
E8            1     XIUCAI L/AU
E9            2     XIUCEN Y/AU
E10           2     XIUCHENG X/AU
E11           1     XIUCHENG XU/AU
E12           1     XIUCHU S/AU

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E1            1     VASILAKOS PAVLOS J/AU
E2            2     VASILAKOS S S/AU
E3            0 --> VASILAKOS, J/AU
E4            4     VASILAKOU M/AU
E5            1     VASILAKY W/AU
E6            4     VASILANTON M/AU
E7            2     VASILANTONE M/AU
E8            2     VASILANTONE M M/AU
E9           13     VASILANTONE MICHAEL/AU
E10           2     VASILANTONE MICHAEL M/AU
E11           1     VASILARAS D/AU
E12           1     VASILARAS DIMITRIOS/AU

=> d his

     (FILE 'HOME' ENTERED AT 07:07:27 ON 29 OCT 2005)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOTECHDS, BIOSIS,
     SCISEARCH' ENTERED AT 07:07:56 ON 29 OCT 2005
L1           82 S TLR-8
L2           37 S L1 AND AGONIST
L3            0 S L1 AND (DETECTION METHOD)
L4            9 S L2 AND (TEST COMPOUND)
                E GORDEN, K/AU
                E XIU, X/AU
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E VASILAKOS, J/AU

=> d l2 ti abs ibib 1-15

L2 ANSWER 1 OF 37 USPATFULL on STN
TI Methods and products based on oligomerization of stress proteins
AB In one aspect, the invention provides methods for determining the biological activity of heat shock proteins or heat shock protein-peptide complexes based on the ATPase activity or the multimeric structure of the heat shock proteins or heal shock protein-peptide complexes, and methods for screening agents that modulate the biological activity of heat shock proteins or heat shock protein-peptide complexes. In another aspect, the invention provides complexes, compositions and methods for enhancing the immunogenicity of a heat shock protein or a complex comprising a heal shock protein and an antigenic molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2005:254901 USPATFULL			
TITLE:	Methods and products based on oligomerization of stress proteins			
INVENTOR(S):	Zabrecky, James R., Waltham, MA, UNITED STATES Liu, Chuanling, Haverhill, MA, UNITED STATES Monks, Stephen A., Arlington, MA, UNITED STATES Wasserman, Andrew, North Andover, MA, UNITED STATES Srivastava, Pramod K., Avon, CT, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005221395	A1	20051006	
APPLICATION INFO.:	US 2003-506097	A1	20030228	(10)
	WO 2003-US6298		20030228	
			20050314	PCT 371 date
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2003-60361257		20020228	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017, US			
NUMBER OF CLAIMS:	81			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	8 Drawing Page(s)			
LINE COUNT:	5793			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 2 OF 37 USPATFULL on STN
TI Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
AB The invention pertains to murine TLR9 and related TLR9s which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR9. The invention further relates to methods of using such murine and non-murine TLR9 nucleic acids and polypeptides, especially in methods for screening for agonists and antagonists of immunostimulatory CpG nucleic acids. Also included are murine TLR9 inhibitors which inhibit murine TLR9 activity by inhibiting the expression or function of murine TLR9. In a further aspect the present invention pertains to murine TLR7 and murine TLR8, as well as related TLR7 and TLR8 molecules which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR7 and TLR8. Methods are included for screening for ligands of TLR7 and TLR8, as well as for inhibitors

and agonists and antagonists of signaling mediated by TLR7 and TLR8.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2005:208942 USPATFULL
TITLE:	Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
INVENTOR(S):	Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF Lipford, Grayson, Watertown, MA, UNITED STATES Wagner, Hermann, Eching, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):	Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005181422	A1	20050818
APPLICATION INFO.:	US 2005-84777	A1	20050318 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2001-954987, filed on 17 Sep 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-233035P	20000915 (60)
	US 2001-263657P	20010123 (60)
	US 2001-291726P	20010517 (60)
	US 2001-300210P	20010622 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS:	18
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	20 Drawing Page(s)
LINE COUNT:	9366

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 3 OF 37 USPATFULL on STN

TI Methods and compositions for enhancing immune response

AB Methods and compositions for enhancing the immune response to an IRM compound by depositing within a localized tissue region an IRM depot preparation that provides an extended residence time of active IRM within the localized tissue region.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:334273 USPATFULL
TITLE:	Methods and compositions for enhancing immune response
INVENTOR(S):	Miller, Richard L., Maplewood, MN, UNITED STATES Tomai, Mark A., Woodbury, MN, UNITED STATES Kedl, Ross M., Denver, CO, UNITED STATES Zarraga, Isidro Angelo Eleazar, Minneapolis, MN, UNITED STATES Ortiz, Ronnie, Apple Valley, MN, UNITED STATES Stoesz, James D., Inver Grove Heights, MN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004265351	A1	20041230
APPLICATION INFO.:	US 2004-821330	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE

PRIORITY INFORMATION: US 2003-533703P 20031231 (60)
US 2003-462140P 20030410 (60)
US 2003-515256P 20031029 (60)
US 2003-515604P 20031030 (60)
US 2004-545424P 20040218 (60)
US 2004-545542P 20040218 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 959
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 4 OF 37 USPATFULL on STN
TI Use of lectins to promote oligomerization of glycoproteins and antigenic molecules
AB The present invention relates to using lectin or lectin-like molecules to promote oligomerization of a glycoprotein or an immunologically and/or biologically active complex comprising glycoproteins. In particular, the invention provides compositions of a molecular complex comprising lectin molecules and immunologically and/or biologically active molecules. Methods of making such molecular complexes and methods of use of the compositions comprising such molecular complexes for the prevention and treatment of diseases, particularly cancer and infectious diseases, and for eliciting an immune response in a subject, are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:326886 USPATFULL
TITLE: Use of lectins to promote oligomerization of glycoproteins and antigenic molecules
INVENTOR(S): Zabrecky, James R., Waltham, MA, UNITED STATES
Monks, Stephen A., Arlington, MA, UNITED STATES
PATENT ASSIGNEE(S): Antigenics Inc. (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004258705	A1	20041223	
APPLICATION INFO.:	US 2004-789220	A1	20040227	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-450721P	20030228 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017
NUMBER OF CLAIMS: 70
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 5764
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 5 OF 37 USPATFULL on STN
TI Delivery of immune response modifier compounds
AB The present invention provides immune response modifiers (IRMs) associated with (typically, attached to, and preferably, covalently attached to) macromolecular support materials. The IRM compounds in such IRM-support complexes retain biological activity. Such attachment of an IRM to a macromolecular support material provides for the localized biological activity of the IRM.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:326879 USPATFULL
TITLE: Delivery of immune response modifier compounds
INVENTOR(S): Wightman, Paul D., Woodbury, MN, UNITED STATES
Zarraga, Isidro Angelo E., Minneapolis, MN, UNITED STATES
Jing, Naiyong, Woodbury, MN, UNITED STATES
Liu, Jie J., Woodbury, MN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004258698	A1	20041223
APPLICATION INFO.:	US 2004-821335	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-462140P	20030410 (60)
	US 2004-545424P	20040218 (60)
	US 2003-515256P	20031029 (60)
	US 2004-545542P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 51
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 2407
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 6 OF 37 USPATFULL on STN
TI Methods for using compositions comprising heat shock proteins or alpha-2-macroglobulin in the treatment of cancer and infectious disease
AB The present invention relates to methods and compositions for the prevention and treatment of infectious diseases, and cancers. The methods of the invention comprises administering (a) a composition comprising a population of complexes of antigenic proteins or antigenic peptides derived from antigenic cells or viral particles and one or more different heat shock proteins; and (b) a non-heat shock protein and non-alpha-2-macroglobulin-based treatment modality. The population or the protein preparation used to produce the antigenic peptides comprises at least 50% of the different proteins or at least 50 different proteins of the antigenic cells or viral particles. Methods for making antigenic peptides comprise digesting a protein preparation of antigenic cells, a cellular fraction thereof, or of viral particles with one or more proteases, or exposing the protein preparation to ATP, guanidium hydrochloride, and/or acidic conditions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:320575 USPATFULL
TITLE: Methods for using compositions comprising heat shock proteins or alpha-2-macroglobulin in the treatment of cancer and infectious disease
INVENTOR(S): Srivastava, Pramod K., Avon, CT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004253228	A1	20041216
APPLICATION INFO.:	US 2004-784012	A1	20040220 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-449001P	20030220 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017
NUMBER OF CLAIMS: 39
EXEMPLARY CLAIM: 1
LINE COUNT: 4653
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 7 OF 37 USPATFULL on STN
TI Delivery of immune response modifier compounds using metal-containing particulate support materials
AB The present invention provides immune response modifiers (IRMs) on particulate support materials that includes one or more metals, including alloys or complexes thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:260225 USPATFULL
TITLE: Delivery of immune response modifier compounds using metal-containing particulate support materials
INVENTOR(S): Wightman, Paul D., Woodbury, MN, UNITED STATES
Liu, Jie J., Woodbury, MN, UNITED STATES
Jing, Naiyong, Woodbury, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004202720	A1	20041014
APPLICATION INFO.:	US 2004-821319	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-462140P	20030410 (60)
	US 2004-545542P	20040218 (60)
	US 2003-515256P	20031029 (60)
	US 2004-545424P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
LINE COUNT: 1759
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 8 OF 37 USPATFULL on STN
TI Methods and products for enhancing immune responses using imidazoquinoline compounds
AB The invention involves administration of an imidazoquinoline agent in combination with another therapeutic agent. The combination of drugs may be administered in synergistic amounts or in various dosages or at various time schedules. The invention also relates to kits and compositions concerning the combination of drugs. The combinations can be used to enhance ADCC, stimulate immune responses and/or patient and treat certain disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:201378 USPATFULL
TITLE: Methods and products for enhancing immune responses

using imidazoquinoline compounds

INVENTOR(S): Krieg, Arthur M., Wellesley, MA, UNITED STATES
Schetter, Christian, Hilden, GERMANY, FEDERAL REPUBLIC OF
Bratzler, Robert L., Concord, MA, UNITED STATES
Vollmer, Jorg, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF
Jurk, Marion, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF
Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF

PATENT ASSIGNEE(S): University of Iowa Research Foundation, Iowa City, IA, 52242 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003139364	A1	20030724
APPLICATION INFO.:	US 2002-272502	A1	20021015 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-329208P	20011012 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS: 87
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 25 Drawing Page(s)
LINE COUNT: 7027
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 9 OF 37 USPATFULL on STN
TI Methods of maturing plasmacytoid dendritic cells using immune response modifier molecules
AB The present invention relates to methods of maturing plasmacytoid dendrites cells using immune response modifier molecules. The present invention also relates to methods of detecting biological activities of matured plasmacytoid dendritic cells and methods of using mature plasmacytoid dendritic cells for therapeutic or prophylactic purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:194103 USPATFULL
TITLE: Methods of maturing plasmacytoid dendritic cells using immune response modifier molecules
INVENTOR(S): Tomai, Mark A., Woodbury, MN, UNITED STATES
Vasilakos, John P., Woodbury, MN, UNITED STATES
Stolpa, John C., St. Paul, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003133913	A1	20030717
APPLICATION INFO.:	US 2002-229829	A1	20020828 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-316144P	20010830 (60)
	US 2002-370177P	20020405 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 93
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)

LINE COUNT: 2566
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 10 OF 37 USPATFULL on STN
TI Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
AB The invention pertains to murine TLR9 and related TLR9s which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR9. The invention further relates to methods of using such murine and non-murine TLR9 nucleic acids and polypeptides, especially in methods for screening for agonists and antagonists of immunostimulatory CpG nucleic acids. Also included are murine TLR9 inhibitors which inhibit murine TLR9 activity by inhibiting the expression or function of murine TLR9. In a further aspect the present invention pertains to murine TLR7 and murine TLR8, as well as related TLR7 and TLR8 molecules which include murine-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biologically functional variants of the murine TLR7 and TLR8. Methods are included for screening for ligands of TLR7 and TLR8, as well as for inhibitors and agonists and antagonists of signaling mediated by TLR7 and TLR8.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:152857 USPATFULL
TITLE: Process for high throughput screening of CpG-based immuno-**agonist**/antagonist
INVENTOR(S): Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
Lipford, Grayson, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF
Wagner, Hermann, Eching, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003104523	A1	20030605	
	US 6943240	B2	20050913	
APPLICATION INFO.:	US 2001-954987	A1	20010917	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-233035P	20000915 (60)
	US 2001-263657P	20010123 (60)
	US 2001-291726P	20010517 (60)
	US 2001-300210P	20010622 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS: 120
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 6814
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 11 OF 37 DGENE COPYRIGHT 2005 The Thomson Corp on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
AN ACC47807 DNA DGENE
AB The invention relates to obtaining a population of mature dendritic cells. The method involves administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount

effective to mature dendritic cells of the subject, and isolating the mature dendritic cells. The method and another new method are useful for treating a disease, by: (a) contacting an isolated plasmacytoid dendritic cell (pDC) with IRM for inducing pDC to express chemokine receptors; (b) contacting the population of pDC with an antigen associated with the disease; (c) enriching the cell population for cells expressing a high level of a chemokine receptor; and (d) administering the enriched cell population to a patient. A cell population (I) is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., AIDS, in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. A cellular adjuvant is useful for provoking an anti-tumour immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation. Sequences ACC47806-07 represent primers for GAPDH gene used in a real-time RT-PCR reaction.

ACCESSION NUMBER: ACC47807 DNA DGENE
TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
INVENTOR: Tomai M A; Vasilakos J P; Stolpa J C
PATENT ASSIGNEE: (MINN)3M INNOVATIVE PROPERTIES CO.
PATENT INFO: WO 2003020889 A2 20030313 84
APPLICATION INFO: WO 2002-US27393 20020828
PRIORITY INFO: US 2001-316144P 20010830
US 2002-370177P 20020405
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2003-393260 [37]
DESCRIPTION: GAPDH gene analysing reverse primer.

L2 ANSWER 12 OF 37 DGENE COPYRIGHT 2005 The Thomson Corp on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
AN ACC47806 DNA DGENE
AB The invention relates to obtaining a population of mature dendritic cells. The method involves administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells. The method and another new method are useful for treating a disease, by: (a) contacting an isolated plasmacytoid dendritic cell (pDC) with IRM for inducing pDC to express chemokine receptors; (b) contacting the population of pDC with an antigen associated with the disease; (c) enriching the cell population for cells expressing a high level of a chemokine receptor; and (d) administering the enriched cell population to a patient. A cell population (I) is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating

e.g., AIDS, in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. A cellular adjuvant is useful for provoking an anti-tumour immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation. Sequences ACC47806-07 represent primers for GAPDH gene used in a real-time RT-PCR reaction.

ACCESSION NUMBER: ACC47806 DNA DGENE
TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
INVENTOR: Tomai M A; Vasilakos J P; Stolpa J C
PATENT ASSIGNEE: (MINN)3M INNOVATIVE PROPERTIES CO.
PATENT INFO: WO 2003020889 A2 20030313 84
APPLICATION INFO: WO 2002-US27393 20020828
PRIORITY INFO: US 2001-316144P 20010830
US 2002-370177P 20020405
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2003-393260 [37]
DESCRIPTION: GAPDH gene analysing forward primer.

L2 ANSWER 13 OF 37 DGENE COPYRIGHT 2005 The Thomson Corp on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
AN ACC47805 DNA DGENE
AB The invention relates to obtaining a population of mature dendritic cells. The method involves administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells. The method and another new method are useful for treating a disease, by: (a) contacting an isolated plasmacytoid dendritic cell (pDC) with IRM for inducing pDC to express chemokine receptors; (b) contacting the population of pDC with an antigen associated with the disease; (c) enriching the cell population for cells expressing a high level of a chemokine receptor; and (d) administering the enriched cell population to a patient. A cell population (I) is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., AIDS, in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. A cellular adjuvant is useful for provoking an anti-tumour immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis,

parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation. Sequences ACC47804-05 represent primers for MIP-3alpha gene used in a real-time RT-PCR reaction.

ACCESSION NUMBER: ACC47805 DNA DGENE
TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
INVENTOR: Tomai M A; Vasilakos J P; Stolpa J C
PATENT ASSIGNEE: (MINN)3M INNOVATIVE PROPERTIES CO.
PATENT INFO: WO 2003020889 A2 20030313 84
APPLICATION INFO: WO 2002-US27393 20020828
PRIORITY INFO: US 2001-316144P 20010830
US 2002-370177P 20020405
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2003-393260 [37]
DESCRIPTION: MIP-3alpha gene analysing reverse primer.

L2 ANSWER 14 OF 37 DGENE COPYRIGHT 2005 The Thomson Corp on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
AN ACC47804 DNA DGENE
AB The invention relates to obtaining a population of mature dendritic cells. The method involves administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells. The method and another new method are useful for treating a disease, by: (a) contacting an isolated plasmacytoid dendritic cell (pDC) with IRM for inducing pDC to express chemokine receptors; (b) contacting the population of pDC with an antigen associated with the disease; (c) enriching the cell population for cells expressing a high level of a chemokine receptor; and (d) administering the enriched cell population to a patient. A cell population (I) is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., AIDS, in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. A cellular adjuvant is useful for provoking an anti-tumour immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation. Sequences ACC47804-05 represent primers for MIP-3alpha gene used in a real-time RT-PCR reaction.

ACCESSION NUMBER: ACC47804 DNA DGENE
TITLE: Obtaining a population of mature dendritic cells, useful for

treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
INVENTOR: Tomai M A; Vasilakos J P; Stolpa J C
PATENT ASSIGNEE: (MINN)3M INNOVATIVE PROPERTIES CO.
PATENT INFO: WO 2003020889 A2 20030313 84
APPLICATION INFO: WO 2002-US27393 20020828
PRIORITY INFO: US 2001-316144P 20010830
US 2002-370177P 20020405
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2003-393260 [37]
DESCRIPTION: MIP-3alpha gene analysing forward primer.

L2 ANSWER 15 OF 37 DGENE COPYRIGHT 2005 The Thomson Corp on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
AN ACC47803 DNA DGENE
AB The invention relates to obtaining a population of mature dendritic cells. The method involves administering an immune response modifier molecule (IRM) that is an **agonist** of Toll-like receptor (TLR)-6, TLR-7, or **TLR-8** to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells. The method and another new method are useful for treating a disease, by: (a) contacting an isolated plasmacytoid dendritic cell (pDC) with IRM for inducing pDC to express chemokine receptors; (b) contacting the population of pDC with an antigen associated with the disease; (c) enriching the cell population for cells expressing a high level of a chemokine receptor; and (d) administering the enriched cell population to a patient. A cell population (I) is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., AIDS, in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. A cellular adjuvant is useful for provoking an anti-tumour immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation. Sequences ACC47802-03 represent primers for MIP-1alpha gene used in a real-time RT-PCR reaction.
ACCESSION NUMBER: ACC47803 DNA DGENE
TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an **agonist** of a Toll-like receptor to a subject -
INVENTOR: Tomai M A; Vasilakos J P; Stolpa J C
PATENT ASSIGNEE: (MINN)3M INNOVATIVE PROPERTIES CO.
PATENT INFO: WO 2003020889 A2 20030313 84
APPLICATION INFO: WO 2002-US27393 20020828
PRIORITY INFO: US 2001-316144P 20010830
US 2002-370177P 20020405
DOCUMENT TYPE: Patent

LANGUAGE: English
OTHER SOURCE: 2003-393260 [37]
DESCRIPTION: MIP-1alpha gene analysing reverse primer.

Refine Search

Search Results -

Terms	Documents
"TLR8"	1

Database:
US Pre-Grant Publication Full-Text Database
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☐ 1. Document ID: US 6943240 B2

L7: Entry 1 of 1 File: USPT Sep 13, 2005

US-PAT-NO: 6943240
DOCUMENT-IDENTIFIER: US 6943240 B2

TITLE: Nucleic acids for high throughput screening of CpG-based immuno-agonist/antagonist

DATE-ISSUED: September 13, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bauer; Stefan	Munich			DE
Lipford; Grayson	Dusseldorf			DE
Wagner; Hermann	Eching			DE

US-CL-CURRENT: 536/23.1; 435/320.1, 435/325

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

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☐ 1. Document ID: US 6558951 B1

L3: Entry 1 of 3 File: USPT May 6, 2003

US-PAT-NO: 6558951
DOCUMENT-IDENTIFIER: US 6558951 B1
** See image for Certificate of Correction **

TITLE: Maturation of dendritic cells with immune response modifying compounds

DATE-ISSUED: May 6, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Tomai; Mark A.	Oakdale	MN		
Vasilakos; John P.	Woodbury	MN		
Ahonen; Cory L.	Hanover	NH		

US-CL-CURRENT: 435/377; 435/325, 435/375, 435/384, 514/291, 546/82

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 4334888 A

L3: Entry 2 of 3 File: USPT Jun 15, 1982

US-PAT-NO: 4334888
DOCUMENT-IDENTIFIER: US 4334888 A

TITLE: Coal desulfurization

DATE-ISSUED: June 15, 1982

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Corcoran; William H.	San Gabriel	CA		
Vasilakos; Nicholas P.	Austin	TX		
Lawson; Daniel D.	Arcadia	CA		

US-CL-CURRENT: 44/622; 201/17, 208/401, 208/435

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 3. Document ID: US 4325707 A

L3: Entry 3 of 3 File: USPT Apr 20, 1982

US-PAT-NO: 4325707
DOCUMENT-IDENTIFIER: US 4325707 A

TITLE: Coal desulfurization by aqueous chlorination

DATE-ISSUED: April 20, 1982

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kalvinskas; John J.	South Pasadena	CA		
Vasilakos; Nick	Pasadena	CA		
Corcoran; William H.	San Gabriel	CA		
Grohmann; Karel	San Dimas	CA		
Rohatgi; Naresh K.	West Covina	CA		

US-CL-CURRENT: 44/625; 201/17

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Neutrophil activation by immune response modifier compounds patent
Generally, the method includes administering a **TLR8**-selective **agonist** and/or ...
required to **identify** a compound as being an **agonist** or a non-**agonist** of a ...
www.freshpatents.com/ Neutrophil-activation-by-immune-response-modifier-compounds-dt20050505ptan2005009625... - 26k - Cached - Similar pages

Science – Diebold et al. 303 (5663): 1529
To **identify** this pathway, we first purified plasmacytoid CD11clow Ly6C+ DC from
... Because responses to some TLR7 and **TLR8** agonists also require endosomal ...
www.sciencemag.org/cgi/content/full/303/5663/1529 - Similar pages

Nucleic acids for high throughput screening of CpG-based immuno ...
Yeast two-hybrid screening methods also may be used to **identify** polypeptides ...
In other embodiments an ISNA **agonist** will bind to a site on TLR7, **TLR8**, ...
www.freepatentsonline.com/6943240.html - 513k - Cached - Similar pages

Blackwell Synergy: Immunology, Vol 114, Issue 4, pp. 507-521 ...
... upstream exons (I and II) and 5'-RACE was used to **identify** this sequence in
... In humans, TLR7 and **TLR8** have been shown to exhibit differential **agonist** ...
www.blackwell-synergy.com/ doi/abs/10.1111/j.1365-2567.2005.02125.x - Similar pages

New Toll-like Receptor Drug Actilon for HCV Therapy
Actilon is a synthetic oligonucleotide and selective TLR9 **agonist** which enhances
... We believe that RNA molecules designed to stimulate TLR7 or **TLR8** could ...
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=> s (resiquimod or R848)
L1          273 (RESIQUIMOD OR R848)

=> s l1 and (TLR8 or toll-like receptor-8)
L2           49 L1 AND (TLR8 OR TOLL-LIKE RECEPTOR-8)

=> s l2 and (agonist)
L3           25 L2 AND (AGONIST)

=> d l3 ti abs ibib tot

L3   ANSWER 1 OF 25     MEDLINE on STN
TI   TLR7/8-mediated activation of human NK cells results in accessory
     cell-dependent IFN-gamma production.
AB   NK cells express receptors that allow them to recognize pathogens and
     activate effector functions such as cytotoxicity and cytokine production.
     Among these receptors are the recently identified TLRs that recognize
     conserved pathogen structures and initiate innate immune responses. We
     demonstrate that human NK cells express TLR3, TLR7, and TLR8 and
     that these receptors are functional. TLR3 is expressed at the cell
     surface where it functions as a receptor for polyinosinic acid:cytidylic
     acid (poly(I:C)) in a lysosomal-independent manner. TLR7/8 signaling is
     sensitive to chloroquine inhibition, indicating a requirement for
     lysosomal signaling as for other cell types. Both R848, an
     agonist of human TLR7 and TLR8, and poly(I:C) activate
     NK cell cytotoxicity against Daudi target cells. However, IFN-gamma
     production is differentially regulated by these TLR agonists. In contrast
     to poly(I:C), R848 stimulates significant IFN-gamma production
     by NK cells. This is accessory cell dependent and is inhibited by
     addition of a neutralizing anti-IL-12 Ab. Moreover, stimulation of
     purified monocyte populations with R848 results in IL-12
     production, and reconstitution of purified NK cells with monocytes results
     in increased IFN-gamma production in response to R848. In
     addition, we demonstrate that while resting NK cells do not transduce
     signals directly in response to R848, they can be primed to do
     so by prior exposure to either IL-2 or IFN-alpha. Therefore, although NK
     cells can be directly activated by TLRs, accessory cells play an important
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and sometimes essential role in the activation of effector functions such as IFN-gamma production and cytotoxicity.

ACCESSION NUMBER: 2005376845 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16034103
TITLE: TLR7/8-mediated activation of human NK cells results in accessory cell-dependent IFN-gamma production.
AUTHOR: Hart Orla M; Athie-Morales Veronica; O'Connor Geraldine M; Gardiner Clair M
CORPORATE SOURCE: Department of Biochemistry, Trinity College, Dublin, Ireland.
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2005 Aug 1) 175 (3) 1636-42.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200510
ENTRY DATE: Entered STN: 20050722
Last Updated on STN: 20051027
Entered Medline: 20051026

L3 ANSWER 2 OF 25 MEDLINE on STN
TI Identification and characterization of a functional, alternatively spliced Toll-like receptor 7 (TLR7) and genomic disruption of **TLR8** in chickens.
AB Based upon the recognition of antiviral compounds and single stranded viral RNA the Toll-like receptors TLR7 and **TLR8** are suggested to play a significant role in initiating antiviral immune responses. Here we report the molecular characterization of the chicken TLR7/8 loci which revealed an intact TLR7 gene and fragments of a **TLR8**-like gene with a 6-kilobase insertion containing chicken repeat 1 (CR1) retroviral-like insertion elements. The chicken TLR7 gene encodes a 1047-amino-acid protein with 62% identity to human TLR7 and a conserved pattern of predicted leucine-rich repeats. Highest levels of chicken TLR7 mRNA were detected in immune-related tissues and cells, especially the spleen, caecal, tonsil and splenic B cells. Alternative spliced forms of TLR7 mRNA were identified in chicken, mouse and human and expressed in similar tissues and cell types to the major form of chicken TLR7. The chicken TLR7+ HD11 cell line and fresh splenocytes produced elevated levels of interleukin-1beta (IL-1beta) mRNA after exposure to the agonists **R848** and loxoribine. Interestingly, none of the TLR7 agonists stimulated increased type I interferon (IFN) mRNA whereas poly(I:C) (a TLR3 **agonist**) up-regulated both chicken IFN-alpha and chicken IFN-beta mRNA. In contrast, TLR7 agonists, particularly **R848** and poly(U) stimulated up-regulation of chicken IL-1beta, and chicken IL-8 mRNAs more effectively than poly(I:C). Stimulation of chicken TLR7 with **R848** was chloroquine sensitive, suggesting signalling within an endosomal compartment, as for mammalian TLR7. The deletion of **TLR8** in galliforms, accompanied with the differential response after exposure to TLR7 agonists, offers insight into the evolution of vertebrate TLR function.

ACCESSION NUMBER: 2005172899 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15804288
TITLE: Identification and characterization of a functional, alternatively spliced Toll-like receptor 7 (TLR7) and genomic disruption of **TLR8** in chickens.
AUTHOR: Philbin Victoria J; Iqbal Muhammad; Boyd Yvonne; Goodchild Marianne J; Beal Richard K; Bumstead Nat; Young John; Smith Adrian L
CORPORATE SOURCE: Division of Immunology and Pathology, Compton Laboratory, Institute of Animal Health, Compton, Newbury, Berkshire, United Kingdom.

SOURCE: Immunology, (2005 Apr) 114 (4) 507-21.
Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 20050405
Last Updated on STN: 20050426
Entered Medline: 20050425

L3 ANSWER 3 OF 25 MEDLINE on STN

TI Randomized, single-blind, placebo-controlled study of topical application of the immune response modulator **resiquimod** in healthy adults.

AB **Resiquimod** is a Toll-like receptor 7 (TLR7) and **TLR8 agonist** that is a potent inducer of alpha interferon (IFN-alpha) and other cytokines. The effects of multiple applications of **resiquimod** gel were assessed in a randomized, single-blind, dose-ranging, placebo-controlled study with 41 healthy subjects. Over a 3-week period, 1-g doses of **resiquimod** or vehicle gel (3:1 randomization) were applied to a 50-cm2 area of the upper arm according to the following regimens: 0.25% applied for 8 h two times per week, 0.05% applied for 8 h two times per week, 0.05% applied for 8 h three times per week, and 0.01% applied for 24 h three times per week. Skin biopsy specimens were obtained prior to the application of the first dose and after the completion of application of the last dose. Dosing with 0.01 and 0.05% **resiquimod** was well tolerated, but that with 0.25% was not; a dose-response relationship for local adverse effects was observed. The level of systemic exposure during multiple topical dosings was <1% of the applied dose. A significant increase in responders after completion of application of the last dose was observed for serum IFN and the interleukin-1 (IL-1) receptor antagonist ($P<0.01$, Fisher's exact test). Increased levels of mRNA for IL-6, IL-8, IFN-alpha, and Mx (an IFN-alpha-inducible protein) were seen in posttreatment biopsy specimens from the group receiving 0.25% **resiquimod** compared to the levels seen in specimens from the group receiving vehicle only ($P<0.01$, Wilcoxon rank sum test). A dose-response-related increase in CD3-positive cells consistent with T-lymphocyte infiltration and a decrease in CD1a-positive cells, consistent with emigration of Langerhans' cells, were observed in treated skin. This study suggests that **resiquimod** is a potent topically active immune response modifier that significantly enhances the cutaneous immune response.

ACCESSION NUMBER: 2003556531 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14638493
TITLE: Randomized, single-blind, placebo-controlled study of topical application of the immune response modulator **resiquimod** in healthy adults.
AUTHOR: Sauder Daniel N; Smith Michael H; Senta-McMillian Therese; Soria Inmaculada; Meng Tze-Chiang
CORPORATE SOURCE: Department of Dermatology, University of Toronto School of Medicine, Toronto, Ontario, Canada.
SOURCE: Antimicrobial agents and chemotherapy, (2003 Dec) 47 (12) 3846-52.
Journal code: 0315061. ISSN: 0066-4804.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031126
Last Updated on STN: 20040114

Entered Medline: 20040113

L3 ANSWER 4 OF 25 USPATFULL on STN
TI Sequence requirements for inhibitory oligonucleotides
AB Novel oligonucleotides having immune inhibitory effects, and methods for their use, are provided. The inhibitory oligonucleotides include those that specifically inhibit certain Toll-like receptors, including TLR7, **TLR8**, and TLR9. Certain of the immunoinhibitory oligonucleotides inhibit a combination of TLRs selected from TLR7, **TLR8**, and TLR9. Inhibitors of TLR9 are characterized by a 5' CC dinucleotide appropriately spaced upstream of a G-rich oligomer. Inhibitors of **TLR8** include specific simple dinucleotides and oligonucleotides ending at their 3' termini with the specific dinucleotides. TLR7 inhibitors include oligonucleotides having a phosphorothioate backbone. Also provided are combinations and conjugates involving the inhibitory oligonucleotides of the invention and other agents, where the other agents include TLR agonists and antigens. Compositions of the invention can be used to shape an immune response, reduce unwanted specific TLR-mediated immunostimulation, and to treat conditions including allergy, asthma, infection, and cancer.

ACCESSION NUMBER: 2005:275170 USPATFULL
TITLE: Sequence requirements for inhibitory oligonucleotides
INVENTOR(S): Jurk, Marion, Duesseldorf, GERMANY, FEDERAL REPUBLIC OF
Vollmer, Jorg, Duesseldorf, GERMANY, FEDERAL REPUBLIC OF
Krieg, Arthur M., Wellesley, MA, UNITED STATES
Uhlmann, Eugen, Glashuetten, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)
Coley Pharmaceutical Group, Inc., Wellesley, MA, UNITED STATES (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005239733	A1	20051027	
APPLICATION INFO.:	US 2004-977560	A1	20041029	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-516221P	20031031	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 3753

L3 ANSWER 5 OF 25 USPATFULL on STN
TI Nonhuman model animal unresponsive to immunopotentiating synthetic compound
AB The present invention relates to provide a non-human animal model unresponsive to a synthetic compound wherein a gene function encoding TLR7 that recognizes an immunopotentiating synthetic compound such as imidazoquinoline lacks on is genomic locus. Whole or part of a gene fragment of a gene site including an intracellular region and a transmembrane region of a TLR7 gene obtained from a mouse gene library is replaced by a plasmid including poly A signal and a marker gene to construct a targeting vector. Then, this targeting vector is linearized and transferred into embryonic stem cells. The target embryonic stem

cells wherein the TLR7 gene function is deleted are microinjected into a mouse blastocyst to generate a chimeric mouse. Then, this chimeric mouse is crossed with a wild-type mouse to generate a heterozygote mouse. Next, the heterozygote mice are intercrossed to obtain a TLR7 knockout mouse.

ACCESSION NUMBER: 2005:270052 USPATFULL
TITLE: Nonhuman model animal unresponsive to immunopotentiating synthetic compound
INVENTOR(S): Akira, Shizuo, Osaka, JAPAN
Tomizawa, Hideyuki, Saitama, JAPAN
Yamaoka, Takashi, Hyogo, JAPAN

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005235372	A1	20051020	
APPLICATION INFO.:	US 2003-496501	A1	20021122	(10)
	WO 2002-JP12234		20021122	
			20040728	PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	JP 2003-2001358295	20011122

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY AND LARDNER, SUITE 500, 3000 K STREET NW, WASHINGTON, DC, 20007, US
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1144

L3 ANSWER 6 OF 25 USPATFULL on STN
TI Toll-like receptor assays
AB Methods of identifying compounds that modulate the interaction between a TLR and a molecule that interacts with the TLR by direct binding or by inclusion in a complex that associates with the TLR are described. Methods of identifying molecules that interact with a TLR are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:240470 USPATFULL
TITLE: Toll-like receptor assays
INVENTOR(S): Latz, Eicke, Boston, MA, UNITED STATES
Visintin, Alberto, Worcester, MA, UNITED STATES
Golenbock, Douglas T., Wellesley, MA, UNITED STATES
PATENT ASSIGNEE(S): University of Massachusetts, Boston, MA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005208470	A1	20050922	
APPLICATION INFO.:	US 2004-14351	A1	20041216	(11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-530115P	20031216 (60)
	US 2003-530699P	20031216 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, P.O. BOX 1022, MINNEAPOLIS, MN, 55440-1022, US
NUMBER OF CLAIMS: 23

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 1593
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 7 OF 25 USPATFULL on STN
TI Immunogenic compositions and methods of use thereof
AB The present invention provides an immunogenic composition comprising lethally irradiated bacteria formulated for mucosal delivery. The present invention further provides methods of preparing a subject immunogenic composition. The present invention further provides a method of inducing an immune response in an individual to an antigen, the method generally involving administering a subject immunogenic composition to a mucosal tissue of the individual.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:202212 USPATFULL
TITLE: Immunogenic compositions and methods of use thereof
INVENTOR(S): Raz, Eyal, Del Mar, CA, UNITED STATES
Fierer, Joshua, LaJolla, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005175630	A1	20050811	
APPLICATION INFO.:	US 2004-21821	A1	20041222	(11)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2004-564913P	20040422	(60)
	US 2003-532786P	20031223	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BOZICEVIC, FIELD & FRANCIS LLP, 1900 UNIVERSITY AVENUE, SUITE 200, EAST PALO ALTO, CA, 94303, US
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3646
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 8 OF 25 USPATFULL on STN
TI TRIF-related adaptor molecule (TRAM) and uses thereof
AB A Toll-IL-1-resistance (TIR) domain-containing adaptor-inducing IFN-β (TRIF)-related adaptor molecule (TRAM) has been identified. TRAM acts specifically in the TLR4 signaling pathway. The invention includes compounds useful for modulating TLR signaling by modulating the effects of TRAM.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:183412 USPATFULL
TITLE: TRIF-related adaptor molecule (TRAM) and uses thereof
INVENTOR(S): Fitzgerald, Katherine A., Cambridge, MA, UNITED STATES
Rowe, Daniel C., Walpole, MA, UNITED STATES
Golenbock, Douglas T., Wellesley, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005158799	A1	20050721	
APPLICATION INFO.:	US 2004-968598	A1	20041018	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-512364P	20031017	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 3447
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 9 OF 25 USPATFULL on STN
TI Small molecule toll-like receptor (TLR) antagonists
AB The invention provides methods and compositions useful for modulating signaling through Toll-like receptors. The methods involve contacting a TLR-expressing cell with a small molecule having a core structure including at least two rings. Certain of the compounds are 4-primary amino quinolines. Many of the compounds and methods are useful specifically for inhibiting immune stimulation involving at least one of TLR9, **TLR8**, TLR7, and TLR3. The methods may have use in the treatment of autoimmunity, inflammation, allergy, asthma, graft rejection, graft versus host disease, infection, sepsis, cancer, and immunodeficiency.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:138623 USPATFULL
TITLE: Small molecule toll-like receptor (TLR) antagonists
INVENTOR(S): Lipford, Grayson B., Watertown, MA, UNITED STATES
Forsbach, Alexandra, Ratingen, GERMANY, FEDERAL REPUBLIC OF
Zepp, Charles, Hardwick, MA, UNITED STATES
PATENT ASSIGNEE(S): Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (U.S. corporation)
Coley Pharmaceutical Group, Inc., Wellesley, MA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005119273	A1	20050602	
APPLICATION INFO.:	US 2004-872196	A1	20040618	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-480588P	20030620	(60)
	US 2004-556007P	20040323	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Alan W. Steele, M.D., Ph.D., Wolf, Greenfield & Sacks, P.C., 600 Atlantic Avenue, Boston, MA, 02210, US
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1-30
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 4382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 10 OF 25 USPATFULL on STN
TI Cell-free methods for identifying compounds that affect toll-like receptor 9 (TLR9) signaling
AB The invention is directed to methods for screening for a compound that affects interaction between a Toll-like receptor (TLR) and a ligand for the TLR. The methods involve direct measurement of interaction using, for example, surface plasmon resonance (SPR), particularly under conditions of pH that mimic those of the TLR in vivo. Compounds identified using the methods of the invention may be useful in the

development of agents useful in the treatment of conditions characterized by undesirable immune activation, e.g., autoimmunity, inflammation, allergy, asthma, and transplantation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2005:117716 USPATFULL
TITLE:	Cell-free methods for identifying compounds that affect toll-like receptor 9 (TLR9) signaling
INVENTOR(S):	Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF Lipford, Grayson, Watertown, MA, UNITED STATES Wagner, Hermann, Eching, GERMANY, FEDERAL REPUBLIC OF Rutz, Mark, Muenchen, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):	Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation) Coley Pharmaceutical Group, Inc., Wellesley, MA, UNITED STATES (non-U.S. corporation) Technische Universitat Munchen, Muenchen, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005100983	A1	20050512	
APPLICATION INFO.:	US 2004-982193	A1	20041105	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-517804P	20031106	(60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS:	16
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	3 Drawing Page(s)
LINE COUNT:	835

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 11 OF 25 USPATFULL on STN

TI Methods and compositions for enhancing immune response

AB Methods and compositions for enhancing the immune response to an IRM compound by depositing within a localized tissue region an IRM depot preparation that provides an extended residence time of active IRM within the localized tissue region.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:334273 USPATFULL
TITLE:	Methods and compositions for enhancing immune response
INVENTOR(S):	Miller, Richard L., Maplewood, MN, UNITED STATES Tomai, Mark A., Woodbury, MN, UNITED STATES Kedl, Ross M., Denver, CO, UNITED STATES Zarraga, Isidro Angelo Eleazar, Minneapolis, MN, UNITED STATES Ortiz, Ronnie, Apple Valley, MN, UNITED STATES Stoesz, James D., Inver Grove Heights, MN, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004265351	A1	20041230	
APPLICATION INFO.:	US 2004-821330	A1	20040409	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING			

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-533703P	20031231 (60)
	US 2003-462140P	20030410 (60)
	US 2003-515256P	20031029 (60)
	US 2003-515604P	20031030 (60)
	US 2004-545424P	20040218 (60)
	US 2004-545542P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 959
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 12 OF 25 USPATFULL on STN
TI Delivery of immune response modifier compounds
AB The present invention provides immune response modifiers (IRMs) associated with (typically, attached to, and preferably, covalently attached to) macromolecular support materials. The IRM compounds in such IRM-support complexes retain biological activity. Such attachment of an IRM to a macromolecular support material provides for the localized biological activity of the IRM.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:326879 USPATFULL
TITLE: Delivery of immune response modifier compounds
INVENTOR(S): Wightman, Paul D., Woodbury, MN, UNITED STATES
Zarraga, Isidro Angelo E., Minneapolis, MN, UNITED STATES
Jing, Naiyong, Woodbury, MN, UNITED STATES
Liu, Jie J., Woodbury, MN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004258698	A1	20041223
APPLICATION INFO.:	US 2004-821335	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-462140P	20030410 (60)
	US 2004-545424P	20040218 (60)
	US 2003-515256P	20031029 (60)
	US 2004-545542P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 51
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 2407
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 13 OF 25 USPATFULL on STN
TI Methods of treating pulmonary fibrotic disorders
AB The present invention provides methods of treating airway remodeling, the methods generally involve administering an effective amount of a

Toll-like receptor **agonist** to an individual suffering from airway remodeling. The present invention provides methods of treating pulmonary fibrosis, the methods generally involving administering an effective amount of a Toll-like receptor **agonist** to an individual in need thereof. The present invention further provides pharmaceutical compositions comprising a TLR **agonist** and a formulation suitable for delivery by inhalation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:315161 USPATFULL			
TITLE:	Methods of treating pulmonary fibrotic disorders			
INVENTOR(S):	Raz, Eyal, Del Mar, CA, UNITED STATES Broide, David, San Diego, CA, UNITED STATES Takabayashi, Kenji, San Diego, CA, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004248837	A1	20041209	
APPLICATION INFO.:	US 2003-697817	A1	20031029	(10)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2002-423035P		20021101	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BOZICEVIC, FIELD & FRANCIS LLP, 1900 UNIVERSITY AVE, SUITE 200, EAST PALO ALTO, CA, 94303			
NUMBER OF CLAIMS:	23			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	6 Drawing Page(s)			
LINE COUNT:	2304			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 14 OF 25 USPATFULL on STN

TI Delivery of immune response modifier compounds using metal-containing particulate support materials

AB The present invention provides immune response modifiers (IRMs) on particulate support materials that includes one or more metals, including alloys or complexes thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:260225 USPATFULL			
TITLE:	Delivery of immune response modifier compounds using metal-containing particulate support materials			
INVENTOR(S):	Wightman, Paul D., Woodbury, MN, UNITED STATES Liu, Jie J., Woodbury, MN, UNITED STATES Jing, Naiyong, Woodbury, MN, UNITED STATES			
PATENT ASSIGNEE(S):	3M Innovative Properties Company (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004202720	A1	20041014	
APPLICATION INFO.:	US 2004-821319	A1	20040409	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING			
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2003-462140P		20030410	(60)
	US 2004-545542P		20040218	(60)
	US 2003-515256P		20031029	(60)
	US 2004-545424P		20040218	(60)
DOCUMENT TYPE:	Utility			

FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
LINE COUNT: 1759
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 15 OF 25 USPATFULL on STN
TI Selective activation of cellular activities mediated through a common toll-like receptor
AB Methods of identifying compounds that selectively modulate cellular activities mediated by a common TLR are provided. Generally, the methods include providing an assay to detect modulation of a first cellular activity mediated by a TLR; providing an assay to detect modulation of a second cellular activity mediated by the TLR; performing each assay using a test compound; and identifying the test compound as a compound that selectively modulates at least one cellular activity of a plurality of activities mediated by a common TLR if the test compound modulates the first cellular activity to a different extent than it modulates the second TLR-mediated cellular activity. Compounds identified by such methods, pharmaceutical compositions including such compounds, and methods of treating a condition by administering such pharmaceutical compositions to a subject are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:247238 USPATFULL
TITLE: Selective activation of cellular activities mediated through a common toll-like receptor
INVENTOR(S): Fink, Jason R., Eagan, MN, UNITED STATES
Gupta, Shalley K., Woodbury, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004191833	A1	20040930	
APPLICATION INFO.:	US 2004-807934	A1	20040324	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-457336P	20030325	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
LINE COUNT: 1382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 16 OF 25 USPATFULL on STN
TI Selective modulation of TLR-mediated biological activity
AB Methods of identifying a compound that selectively modulates at least one TLR-mediated cellular activity are disclosed. Generally, the methods include identifying a compound as a compound that selectively modulates at least one TLR-mediated cellular activity if the compound modulates one TLR-mediated cellular activity to a different extent than it modulates a second TLR-mediated cellular activity. Compounds so identified and pharmaceutical compositions including such compounds are also disclosed. Methods of selectively modulating immune cells and methods of treating certain conditions are also provided. Such methods include administering to cells or a subject a compound that selectively modulates a TLR-mediated cellular activity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:221317 USPATFULL
TITLE: Selective modulation of TLR-mediated biological activity
INVENTOR(S): Fink, Jason R., Eagan, MN, UNITED STATES
Gorden, Keith B., Maplewood, MN, UNITED STATES
Gorski, Kevin S., White Bear Lake, MN, UNITED STATES
Gupta, Shalley K., Woodbury, MN, UNITED STATES
Qiu, Xiaohong, Rosemount, MN, UNITED STATES
Vasilakos, John P., Woodbury, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004171086	A1	20040902
APPLICATION INFO.:	US 2004-788731	A1	20040227 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-450484P	20030227 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 1870
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 17 OF 25 USPATFULL on STN
TI Toll-like receptor 3 signaling agonists and antagonists
AB Compositions and methods are provided to identify, characterize, and optimize immunostimulatory compounds, their agonists and antagonists, working through TLR3.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:237844 USPATFULL
TITLE: Toll-like receptor 3 signaling agonists and antagonists
INVENTOR(S): Lipford, Grayson B., Dusseldorf, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003166001	A1	20030904
APPLICATION INFO.:	US 2002-265072	A1	20021005 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-327520P	20011005 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3285
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 18 OF 25 USPATFULL on STN
TI Methods and products for enhancing immune responses using

imidazoquinoline compounds

AB The invention involves administration of an imidazoquinoline agent in combination with another therapeutic agent. The combination of drugs may be administered in synergistic amounts or in various dosages or at various time schedules. The invention also relates to kits and compositions concerning the combination of drugs. The combinations can be used to enhance ADCC, stimulate immune responses and/or patient and treat certain disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:201378 USPATFULL
TITLE:	Methods and products for enhancing immune responses using imidazoquinoline compounds
INVENTOR(S):	Krieg, Arthur M., Wellesley, MA, UNITED STATES Schetter, Christian, Hilden, GERMANY, FEDERAL REPUBLIC OF Bratzler, Robert L., Concord, MA, UNITED STATES Vollmer, Jorg, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF Jurk, Marion, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):	University of Iowa Research Foundation, Iowa City, IA, 52242 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003139364	A1	20030724	
APPLICATION INFO.:	US 2002-272502	A1	20021015	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-329208P	20011012	(60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS:	87
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	25 Drawing Page(s)
LINE COUNT:	7027

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 19 OF 25 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

TI TLR7/8-mediated activation of human NK cells results in accessory cell-dependent IFN-γ production.

AB NK cells express receptors that allow them to recognize pathogens and activate effector functions such as cytotoxicity and cytokine production. Among these receptors are the recently identified TLRs that recognize conserved pathogen structures and initiate innate immune responses. We demonstrate that human NK cells express TLR3, TLR7, and **TLR8** and that these receptors are functional. TLR3 is expressed at the cell surface where it functions as a receptor for polyinosinic acid:cytidylic acid (poly(I:C)) in a lysosomal-independent manner. TLR7/8 signaling is sensitive to chloroquine inhibition, indicating a requirement for lysosomal signaling as for other cell types. Both **R848**, an **agonist** of human TLR7 and **TLR8**, and poly(I:C) activate NK cell cytotoxicity against Daudi target cells. However, IFN-γ production is differentially regulated by these TLR agonists. In contrast to poly(I:C), **R848** stimulates significant IFN-γ production by NK cells. This is accessory cell dependent and is inhibited by addition of a neutralizing anti-IL-12 Ab. Moreover, stimulation of purified monocyte populations with **R848** results in IL-12 production, and reconstitution of purified NK cells with monocytes results

in increased IFN-γ production in response to **R848**. In addition, we demonstrate that while resting NK cells do not transduce signals directly in response to **R848**, they can be primed to do so by prior exposure to either IL-2 or IFN-α. Therefore, although NK cells can be directly activated by TLRs, accessory cells play an important and sometimes essential role in the activation of effector functions such as IFN-γ production and cytotoxicity. Copyright .COPYRGT. 2005 by The American Association of Immunologists, Inc.

ACCESSION NUMBER: 2005331017 EMBASE
TITLE: TLR7/8-mediated activation of human NK cells results in accessory cell-dependent IFN-γ production.
AUTHOR: Hart O.M.; Athie-Morales V.; O'Connor G.M.; Gardiner C.M.
CORPORATE SOURCE: Dr. C.M. Gardiner, Department of Biochemistry, Trinity College, Dublin 2, Ireland. clair.gardiner@tcd.ie
SOURCE: Journal of Immunology, (1 Aug 2005) Vol. 175, No. 3, pp. 1636-1642.
Refs: 51
ISSN: 0022-1767 CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050825
Last Updated on STN: 20050825

L3 ANSWER 20 OF 25 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

TI Immunization with HIV-1 gag protein conjugated to a TLR7/8 **agonist** results in the generation of HIV-1 gag-specific Th1 and CD8(+) T cell responses.

AB One strategy to induce optimal cellular and humoral immune responses following immunization is to use vaccines or adjuvants that target dendritic cells and B cells. Activation of both cell types can be achieved using specific TLR ligands or agonists directed against their cognate receptor. In this study, we compared the ability of the TLR7/8 **agonist** R-848, which signals only via TLR7 in mice, with CpG oligodeoxynucleotides for their capacity to induce HIV-1 Gag-specific T cell and Ab responses when used as vaccine adjuvants with HIV-1 Gag protein in mice. Injection of R-848 and CpG oligodeoxynucleotides alone enhanced the innate immune responses in vivo as demonstrated by high serum levels of inflammatory cytokines, including IL-12p70 and IFN-α, and increased expression of CD80, CD86, and CD40 on CD11c(+) dendritic cells. By contrast, R-848 was a relatively poor adjuvant for inducing primary Th1 or CD8(+) T cell responses when administered with HIV-1 Gag protein. However, when a TLR7/8 **agonist** structurally and functionally similar to R-848 was conjugated to HIV-1 Gag protein both Th1 and CD8(+) T cells responses were elicited as determined by intracellular cytokine and tetramer staining. Moreover, within the population of HIV-1 Gag-specific CD8(+) CD62(low) cells, .apprx.50% of cells expressed CD127, a marker shown to correlate with the capacity to develop into long-term memory cells. Overall, these data provide evidence that TLR7/8 agonists can be effective vaccine adjuvants for eliciting strong primary immune responses with a viral protein in vivo, provided vaccine delivery is optimized. Copyright .COPYRGT. 2005 by The American Association of Immunologists, Inc.

ACCESSION NUMBER: 2005261964 EMBASE
TITLE: Immunization with HIV-1 gag protein conjugated to a TLR7/8 **agonist** results in the generation of HIV-1 gag-specific Th1 and CD8(+) T cell responses.
AUTHOR: Wille-Reece U.; Wu C.-Y.; Flynn B.J.; Kedl R.M.; Seder R.A.
CORPORATE SOURCE: Dr. R.A. Seder, Cellular Immunology Section, Vaccine Research Center, National Institute of Allergy and

Infectious Diseases, 40 Convent Drive, Bethesda, MD 20892, United States. rseder@mail.nih.gov

SOURCE: Journal of Immunology, (15 Jun 2005) Vol. 174, No. 12, pp. 7676-7683.
Refs: 44
ISSN: 0022-1767 CODEN: JOIMA3

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050707
Last Updated on STN: 20050707

L3 ANSWER 21 OF 25 EMBASE

TI Identification and characterization of a functional, alternatively spliced Toll-like receptor 7 (TLR7) and genomic disruption of **TLR8** in chickens.

AB Based upon the recognition of antiviral compounds and single stranded viral RNA the Toll-like receptors TLR7 and **TLR8** are suggested to play a significant role in initiating antiviral immune responses. Here we report the molecular characterization of the chicken TLR7/8 loci which revealed an intact TLR7 gene and fragments of a **TLR8**-like gene with a 6-kilobase insertion containing chicken repeat 1 (CR1) retroviral-like insertion elements. The chicken TLR7 gene encodes a 1047-amino-acid protein with 62% identity to human TLR7 and a conserved pattern of predicted leucine-rich repeats. Highest levels of chicken TLR7 mRNA were detected in immune-related tissues and cells, especially the spleen, caecal, tonsil and splenic B cells. Alternative spliced forms of TLR7 mRNA were identified in chicken, mouse and human and expressed in similar tissues and cell types to the major form of chicken TLR7. The chicken TLR7 (+) HD11 cell line and fresh splenocytes produced elevated levels of interleukin-1β (IL-1β) mRNA after exposure to the agonists **R848** and loxoribine. Interestingly, none of the TLR7 agonists stimulated increased type I interferon (IFN) mRNA whereas poly(I:C) (a TLR3 **agonist**) up-regulated both chicken IFN-α and chicken IFN-β mRNA. In contrast, TLR7 agonists, particularly **R848** and poly(U) stimulated up-regulation of chicken IL-1β and chicken IL-8 mRNAs more effectively than poly(I:C). Stimulation of chicken TLR7 with **R848** was chloroquine sensitive, suggesting signalling within an endosomal compartment, as for mammalian TLR7. The deletion of **TLR8** in galliforms, accompanied with the differential response after exposure to TLR7 agonists, offers insight into the evolution of vertebrate TLR function. .COPYRGT. 2005 Blackwell Publishing Ltd.

ACCESSION NUMBER: 2005159932 EMBASE
TITLE: Identification and characterization of a functional, alternatively spliced Toll-like receptor 7 (TLR7) and genomic disruption of **TLR8** in chickens.
AUTHOR: Philbin V.J.; Iqbal M.; Boyd Y.; Goodchild M.J.; Beal R.K.; Bumstead N.; Young J.; Smith A.L.
CORPORATE SOURCE: Dr. A.L. Smith, Division of Immunology and Pathology, Institute for Animal Health, Compton Laboratory, Newbury, Berkshire, RG20 7NN, United Kingdom.
adrian.smith@bbsrc.ac.uk
SOURCE: Immunology, (2005) Vol. 114, No. 4, pp. 507-521.
Refs: 66
ISSN: 0019-2805 CODEN: IMMUAM
COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050505
Last Updated on STN: 20050505

L3 ANSWER 22 OF 25 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN
TI Therapeutic targeting of Toll-like receptors.
AB Toll-like receptors (TLRs) play a crucial role in innate immune response in mammals. Individual TLRs recognize microbial components that are conserved among pathogens and activate their signaling pathways. Each TLR has its own cascade of signaling pathway for exhibiting its specific responses through selective utilization of TIR domain-containing adaptors. Increasing evidence for roles of TLRs in various diseases provides us new insights for a basis of new therapies. In this review, we discuss the possibilities of therapeutics targeting TLRs in various diseases and explain potential problems associated with such approaches. .COPYRGT. 2004 Elsevier Ltd. All rights reserved.
ACCESSION NUMBER: 2005065702 EMBASE
TITLE: Therapeutic targeting of Toll-like receptors.
AUTHOR: Uematsu S.; Ishii K.J.; Akira S.
CORPORATE SOURCE: S. Akira, Department of Host Defense, Res. Inst. for Microbial Diseases, Osaka University, 3-1 Yamada-oka, Suita, Osaka 565-0871, Japan. sakira@biken.osaka-u.ac.jp
SOURCE: Drug Discovery Today: Therapeutic Strategies, (2004) Vol. 1, No. 3, pp. 299-304.
Refs: 22
ISSN: 1740-6773
PUBLISHER IDENT.: S 1740-6773(04)00061-0
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050224
Last Updated on STN: 20050224

L3 ANSWER 23 OF 25 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN
TI Human Immunodeficiency Virus Type 1 Activates Plasmacytoid Dendritic Cells and Concomitantly Induces the Bystander Maturation of Myeloid Dendritic Cells.
AB In this study, we analyzed the phenotypic and physiological consequences of the interaction of plasmacytoid dendritic cells (pDCs) with human immunodeficiency virus type 1 (HIV-1). pDCs are one cellular target of HIV-1 and respond to the virus by producing alpha/beta interferon (IFN-α/β) and chemokines. The outcome of this interaction, notably on the function of bystander myeloid DC (CD11c(+) DCs), remains unclear. We therefore evaluated the effects of HIV-1 exposure on these two DC subsets under various conditions. Blood-purified pDCs and CD11c(+) DCs were exposed in vitro to HIV-1, after which maturation markers, cytokine production, migratory capacity, and CD4 T-cell stimulatory capacity were analyzed, pDCs exposed to different strains of infectious or even chemically inactivated, nonreplicating HIV-1 strongly upregulated the expression of maturation markers, such as CD83 and functional CCR7, analogous to exposure to R-848, a synthetic **agonist** of toll-like receptor-7 and -8. In addition, HIV-1-activated pDCs produced cytokines

(IFN-α and tumor necrosis factor alpha), migrated in response to CCL19 and, in coculture, matured CD11c(+) DCs, which are not directly activated by HIV. pDCs also acquired the ability to stimulate naive CD4(+) T cells, albeit less efficiently than CD11c (+) DCs. This HIV-1-induced maturation of both DC subsets may explain their disappearance from the blood of patients with high viral loads and may have important consequences on HIV-1 cellular transmission and HIV-1-specific T-cell responses.

ACCESSION NUMBER: 2004198414 EMBASE
TITLE: Human Immunodeficiency Virus Type 1 Activates Plasmacytoid Dendritic Cells and Concomitantly Induces the Bystander Maturation of Myeloid Dendritic Cells.
AUTHOR: Fonteneau J.-F.; Larsson M.; Beignon A.-S.; McKenna K.; Dasilva I.; Amara A.; Liu Y.-J.; Lifson J.D.; Littman D.R.; Bhardwaj N.
CORPORATE SOURCE: N. Bhardwaj, NYU School of Medicine, Department of Pathology, MSB507, 550 First Ave., New York, NY 10016, France. bhardn02@med.nyu.edu
SOURCE: Journal of Virology, (2004) Vol. 78, No. 10, pp. 5223-5232. Refs: 51
ISSN: 0022-538X CODEN: JOVIAM
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040520
Last Updated on STN: 20040520

L3 ANSWER 24 OF 25 EMBASE

TI Randomized, Single-Blind, Placebo-Controlled Study of Topical Application of the Immune Response Modulator **Resiquimod** in Healthy Adults.

AB **Resiquimod** is a Toll-like receptor 7 (TLR7) and **TLR8 agonist** that is a potent inducer of alpha interferon (IFN-α) and other cytokines. The effects of multiple applications of **resiquimod** gel were assessed in a randomized, single-blind, dose-ranging, placebo-controlled study with 41 healthy subjects. Over a 3-week period, 1-g doses of **resiquimod** or vehicle gel (3:1 randomization) were applied to a 50-cm(2) area of the upper arm according to the following regimens: 0.25% applied for 8 h two times per week, 0.05% applied for 8 h two times per week, 0.05% applied for 8 h three times per week, and 0.01% applied for 24 h three times per week. Skin biopsy specimens were obtained prior to the application of the first dose and after the completion of application of the last dose. Dosing with 0.01 and 0.05% **resiquimod** was well tolerated, but that with 0.25% was not; a dose-response relationship for local adverse effects was observed. The level of systemic exposure during multiple topical dosings was <1% of the applied dose. A significant increase in responders after completion of application of the last dose was observed for serum IFN and the interleukin-1 (IL-1) receptor antagonist ($P < 0.01$, Fisher's exact test). Increased levels of mRNA for IL-6, IL-8, IFN-α, and Mx (an IFN-α-inducible protein) were seen in posttreatment biopsy specimens from the group receiving 0.25% **resiquimod** compared to the levels seen in specimens from the group receiving vehicle only ($P < 0.01$, Wilcoxon rank sum test). A dose-response-related increase in CD3-positive cells consistent with T-lymphocyte infiltration and a decrease in CD1a-positive cells, consistent with emigration of Langerhans' cells, were observed in treated skin. This study suggests that **resiquimod** is a potent topically active immune response modifier that significantly enhances the cutaneous immune response.

ACCESSION NUMBER: 2003493058 EMBASE
TITLE: Randomized, Single-Blind, Placebo-Controlled Study of Topical Application of the Immune Response Modulator **Resiquimod** in Healthy Adults.
AUTHOR: Sauder D.N.; Smith M.H.; Senta-McMillian T.; Soria I.; Meng T.-C.
CORPORATE SOURCE: T.-C. Meng, 3M Pharmaceuticals, 3M Center, Saint Paul, MN 55144-1000, Canada. tmeng1@mmm.com
SOURCE: Antimicrobial Agents and Chemotherapy, (2003) Vol. 47, No. 12, pp. 3846-3852.
Refs: 21
ISSN: 0066-4804 CODEN: AMACCQ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040116
Last Updated on STN: 20040116

L3 ANSWER 25 OF 25 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Stimulating antibody dependent cellular cytotoxicity, modulating immune response and inducing antigen-specific immune response in subject by administering imidazoquinoline agents in conjunction with other agents.
AN 2003-829705 [77] WPIDS
AB US2003139364 A UPAB: 20031128
NOVELTY - Stimulating (M1) antibody dependent cellular cytotoxicity (ADCC), modulating (M2) immune response and inducing (M3) antigen-specific immune response in a subject by administering an antibody, immunostimulatory nucleic acid and antigen and immunostimulatory nucleic acid respectively along with imidazoquinoline agents, is new.

DETAILED DESCRIPTION - Stimulating (M1) antibody dependent cellular cytotoxicity (ADCC) in a subject by administering an antibody and an agent (I) chosen from imidazoquinoline agent (IA) and a C8-substituted guanosine, modulating (M2) immune response in a subject by administering immunostimulatory nucleic acid and (I) and inducing (M3) antigen-specific immune response in a subject by administering an antigen, an (IA) and immunostimulatory nucleic acid.

INDEPENDENT CLAIMS are also included for:

(1) a composition (C1) comprising (IA) and an immunostimulatory nucleic acid;

(2) a composition (C2) comprising an (IA) and an antibody;

(3) a composition (C3) comprising an (IA) and a disorder-specific medicament; and

(4) screening (M4) for comparing Toll-like receptor (TLR) signaling activity of a test compound with TLR signaling activity of IA involves contacting a functional TLR chosen from TLR7 and **TLR8** with a reference (IA) and detecting a reference response mediated by a TLR signal transduction pathway, contacting the functional TLR with a test compound and detecting a test response mediated by a TLR signal transduction pathway and comparing the test response with reference response to compare the TLR signaling activity of the test compound with (IA).

ACTIVITY - Antiasthmatic; Cytostatic; Antimicrobial; Dermatological; Virucide.

MECHANISM OF ACTION - Stimulator of ADCC; Modulator of immune response; Inducer of antigen-specific immune response (claimed); Inducer of expression of cytokines including interferons; Stimulator of Th1 immune response; Inhibitor of production of Th2 cytokines such as IL-4, IL-5 and IL-13; Increase NK cell lytic activity; Stimulator of B cell proliferation

and differentiation.

The activity of R-848 to induce IFN- alpha in monocyte-derived dendritic cells (mDCs) was evaluated as follows: unfractionated human peripheral blood mononuclear cell (PBMC), containing mDCs and plasmacytoid dendritic cells (pDCs), were incubated for 48 hours in the presence of varying concentrations of R-848 (0.01-1.0 micro g/ml), varying concentrations of CpG ODN 2006 (0.2-3.0 mu g/ml), varying concentrations of negative control ODN 5177 (5' TCCGCCCTGTGACATGCATT 3'). Staphylococcal enterotoxin B or media alone, and then the concentration of IFN- alpha in the supernatant was measured by ELISA. R-848 induced higher amounts of IFN- alpha upon incubation of human PBMC than type B CpG ODN 2006.

USE - (M1) is useful for stimulating ADCC in a subject. The antibody is chosen from anti-cancer antibody, anti-viral antibody, anti-bacterial antibody, anti-fungal antibody, anti-allergen antibody and anti-self antigen antibody. The subject has or is at risk of having a disorder chosen from asthma/allergy, infectious disease, cancer and warts. (IA) which is imidazoquinoline amine is administered prior to the antibody. (IA) is chosen from imiquimod/R-837 and S-28463/R-848. (M2) is useful for modulating an immune response in a subject who is an immunocompromised subject or elderly or an infant. The immune response is a Th1 immune response, ADCC, innate immune response, local immune response, mucosal immune response or systemic immune response. The agent is administered prior to the immunostimulatory nucleic acid. The amount is effective to modulate the immune response is a synergistic amount. The method further involves administering disorder-specific medicament chosen from cancer medicament, and asthma/allergy medicament, an infectious disease medicament and a wart medicament to the subject. The cancer medicament is chosen from chemotherapeutic agent, an immunotherapeutic agent and a cancer vaccine. The asthma/allergy medicament is chosen from steroids, immunomodulators, anti-inflammatory agents, bronchodilators, leukotriene modifiers, beta 2 agonists,and anticholinergics. The anti-microbial medicament is chosen from an anti-bacterial agent, an anti-viral agent, an anti-fungal agent, and an anti-parasitic agent. The method further involves exposing the subject to an antigen chosen from tumor antigen, viral antigen, bacterial antigen, parasitic antigen, and fungal antigen and where the immune response is an antigen-specific immune response. The subject has or is at the risk of developing an infectious disease, developing a cancer, or developing asthma/allergy. (M3) is useful for inducing an antigen-specific immune response in a subject (all claimed). (M2) is useful for treating a subject having asthma/allergy, infectious disease, cancer and warts.

ADVANTAGE - IA used in conjunction with other agents such as antibodies, immunostimulatory nucleic acid, antigens, C8-substituted guanosines and disorder-specific medicaments provides improved results.

DESCRIPTION OF DRAWING(S) - The figure shows a bar graph depicting hTLR9-mediated activation of NF-kappa B by CpG ODN 2006, but not by R-848.

Dwg.1/20

ACCESSION NUMBER: 2003-829705 [77] WPIDS
DOC. NO. NON-CPI: N2003-662840
DOC. NO. CPI: C2003-233743

TITLE: Stimulating antibody dependent cellular cytotoxicity, modulating immune response and inducing antigen-specific immune response in subject by administering imidazoquinoline agents in conjunction with other agents.

DERWENT CLASS: B04 B05 D16 S03

INVENTOR(S): BAUER, S; BRATZLER, R L; JURK, M; KRIEG, A M; SCHETTER, C; VOLLMER, J

PATENT ASSIGNEE(S): (IOWA) UNIV IOWA RES FOUND; (COLE-N) COLEY PHARM GMBH; (COLE-N) COLEY PHARM GROUP INC
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

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US 2003139364    A1 20030724 (200377)*      112
WO 2003094836    A2 20031120 (200403)  EN
   RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU
       MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW
    W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
       DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
       KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
       RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZW
AU 2002360278    A1 20031111 (200442)
EP 1478371       A2 20041124 (200477)  EN
    R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC
       MK NL PT RO SE SI SK TR
JP 2005519990    W  20050707 (200545)      158
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APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2003139364	A1 Provisional	US 2001-329208P	20011012
		US 2002-272502	20021015
WO 2003094836	A2	WO 2002-US33051	20021015
AU 2002360278	A1	AU 2002-360278	20021015
EP 1478371	A2	EP 2002-795524	20021015
		WO 2002-US33051	20021015
JP 2005519990	W	WO 2002-US33051	20021015
		JP 2004-502925	20021015

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002360278	A1 Based on	WO 2003094836
EP 1478371	A2 Based on	WO 2003094836
JP 2005519990	W Based on	WO 2003094836

PRIORITY APPLN. INFO: US 2001-329208P 20011012; US 2002-272502 20021015

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=> s (toll-like receptor and agonist)
L4        1301 (TOLL-LIKE RECEPTOR AND AGONIST)

=> s l4 and l1
L5          38 L4 AND L1

=> s l5 and l2
L6          23 L5 AND L2

=> d l6 ti abs ibib tot

L6   ANSWER 1 OF 23     MEDLINE on STN
TI   Identification and characterization of a functional, alternatively spliced
     Toll-like receptor 7 (TLR7) and genomic
     disruption of TLR8 in chickens.
AB   Based upon the recognition of antiviral compounds and single stranded
     viral RNA the Toll-like receptors TLR7 and TLR8 are suggested to
     play a significant role in initiating antiviral immune responses. Here we
     report the molecular characterization of the chicken TLR7/8 loci which
     revealed an intact TLR7 gene and fragments of a TLR8-like gene
     with a 6-kilobase insertion containing chicken repeat 1 (CR1)
     retroviral-like insertion elements. The chicken TLR7 gene encodes a
     1047-amino-acid protein with 62% identity to human TLR7 and a conserved
     pattern of predicted leucine-rich repeats. Highest levels of chicken TLR7
     mRNA were detected in immune-related tissues and cells, especially the
     spleen, caecal, tonsil and splenic B cells. Alternative spliced forms of
     TLR7 mRNA were identified in chicken, mouse and human and expressed in
     similar tissues and cell types to the major form of chicken TLR7. The
     chicken TLR7+ HD11 cell line and fresh splenocytes produced elevated
     levels of interleukin-1beta (IL-1beta) mRNA after exposure to the agonists
     R848 and loxoribine. Interestingly, none of the TLR7 agonists
     stimulated increased type I interferon (IFN) mRNA whereas poly(I:C) (a
     TLR3 agonist) up-regulated both chicken IFN-alpha and chicken
     IFN-beta mRNA. In contrast, TLR7 agonists, particularly R848
     and poly(U) stimulated up-regulation of chicken IL-1beta, and chicken IL-8
     mRNAs more effectively than poly(I:C). Stimulation of chicken TLR7 with
     R848 was chloroquine sensitive, suggesting signalling within an
     endosomal compartment, as for mammalian TLR7. The deletion of
     TLR8 in galliforms, accompanied with the differential response
     after exposure to TLR7 agonists, offers insight into the evolution of
     vertebrate TLR function.
ACCESSION NUMBER:    2005172899     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 15804288
TITLE:               Identification and characterization of a functional,
                     alternatively spliced Toll-like
                     receptor 7 (TLR7) and genomic disruption of
                     TLR8 in chickens.
AUTHOR:              Philbin Victoria J; Iqbal Muhammad; Boyd Yvonne; Goodchild
                     Marianne J; Beal Richard K; Bumstead Nat; Young John; Smith
                     Adrian L
CORPORATE SOURCE:    Division of Immunology and Pathology, Compton Laboratory,
                     Institute of Animal Health, Compton, Newbury, Berkshire,
                     United Kingdom.
SOURCE:              Immunology, (2005 Apr) 114 (4) 507-21.
                     Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY:        England: United Kingdom
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200504
ENTRY DATE:          Entered STN: 20050405
```

Last Updated on STN: 20050426
Entered Medline: 20050425

L6 ANSWER 2 OF 23 MEDLINE on STN
TI Randomized, single-blind, placebo-controlled study of topical application of the immune response modulator **resiquimod** in healthy adults.
AB **Resiquimod** is a **Toll-like receptor** 7 (TLR7) and **TLR8 agonist** that is a potent inducer of alpha interferon (IFN-alpha) and other cytokines. The effects of multiple applications of **resiquimod** gel were assessed in a randomized, single-blind, dose-ranging, placebo-controlled study with 41 healthy subjects. Over a 3-week period, 1-g doses of **resiquimod** or vehicle gel (3:1 randomization) were applied to a 50-cm2 area of the upper arm according to the following regimens: 0.25% applied for 8 h two times per week, 0.05% applied for 8 h two times per week, 0.05% applied for 8 h three times per week, and 0.01% applied for 24 h three times per week. Skin biopsy specimens were obtained prior to the application of the first dose and after the completion of application of the last dose. Dosing with 0.01 and 0.05% **resiquimod** was well tolerated, but that with 0.25% was not; a dose-response relationship for local adverse effects was observed. The level of systemic exposure during multiple topical dosings was <1% of the applied dose. A significant increase in responders after completion of application of the last dose was observed for serum IFN and the interleukin-1 (IL-1) receptor antagonist ($P<0.01$, Fisher's exact test). Increased levels of mRNA for IL-6, IL-8, IFN-alpha, and Mx (an IFN-alpha-inducible protein) were seen in posttreatment biopsy specimens from the group receiving 0.25% **resiquimod** compared to the levels seen in specimens from the group receiving vehicle only ($P<0.01$, Wilcoxon rank sum test). A dose-response-related increase in CD3-positive cells consistent with T-lymphocyte infiltration and a decrease in CD1a-positive cells, consistent with emigration of Langerhans' cells, were observed in treated skin. This study suggests that **resiquimod** is a potent topically active immune response modifier that significantly enhances the cutaneous immune response.
ACCESSION NUMBER: 2003556531 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14638493
TITLE: Randomized, single-blind, placebo-controlled study of topical application of the immune response modulator **resiquimod** in healthy adults.
AUTHOR: Sauder Daniel N; Smith Michael H; Senta-McMillian Therese; Soria Inmaculada; Meng Tze-Chiang
CORPORATE SOURCE: Department of Dermatology, University of Toronto School of Medicine, Toronto, Ontario, Canada.
SOURCE: Antimicrobial agents and chemotherapy, (2003 Dec) 47 (12) 3846-52.
Journal code: 0315061. ISSN: 0066-4804.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031126
Last Updated on STN: 20040114
Entered Medline: 20040113

L6 ANSWER 3 OF 23 USPATFULL on STN
TI Sequence requirements for inhibitory oligonucleotides
AB Novel oligonucleotides having immune inhibitory effects, and methods for their use, are provided. The inhibitory oligonucleotides include those that specifically inhibit certain Toll-like receptors, including TLR7, **TLR8**, and TLR9. Certain of the immunoinhibitory oligonucleotides

inhibit a combination of TLRs selected from TLR7, **TLR8**, and TLR9. Inhibitors of TLR9 are characterized by a 5' CC dinucleotide appropriately spaced upstream of a G-rich oligomer. Inhibitors of **TLR8** include specific simple dinucleotides and oligonucleotides ending at their 3' termini with the specific dinucleotides. TLR7 inhibitors include oligonucleotides having a phosphorothioate backbone. Also provided are combinations and conjugates involving the inhibitory oligonucleotides of the invention and other agents, where the other agents include TLR agonists and antigens. Compositions of the invention can be used to shape an immune response, reduce unwanted specific TLR-mediated immunostimulation, and to treat conditions including allergy, asthma, infection, and cancer.

ACCESSION NUMBER: 2005:275170 USPATFULL
TITLE: Sequence requirements for inhibitory oligonucleotides
INVENTOR(S): Jurk, Marion, Duesseldorf, GERMANY, FEDERAL REPUBLIC OF
Vollmer, Jorg, Duesseldorf, GERMANY, FEDERAL REPUBLIC OF
Krieg, Arthur M., Wellesley, MA, UNITED STATES
Uhlmann, Eugen, Glashuetten, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)
Coley Pharmaceutical Group, Inc., Wellesley, MA, UNITED STATES (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005239733	A1	20051027	
APPLICATION INFO.:	US 2004-977560	A1	20041029	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-516221P	20031031	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 3753

L6 ANSWER 4 OF 23 USPATFULL on STN
TI **Toll-like receptor** assays
AB Methods of identifying compounds that modulate the interaction between a TLR and a molecule that interacts with the TLR by direct binding or by inclusion in a complex that associates with the TLR are described. Methods of identifying molecules that interact with a TLR are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:240470 USPATFULL
TITLE: **Toll-like receptor** assays
INVENTOR(S): Latz, Eicke, Boston, MA, UNITED STATES
Visintin, Alberto, Worcester, MA, UNITED STATES
Golenbock, Douglas T., Wellesley, MA, UNITED STATES
PATENT ASSIGNEE(S): University of Massachusetts, Boston, MA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005208470	A1	20050922

APPLICATION INFO.: US 2004-14351 A1 20041216 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-530115P	20031216 (60)
	US 2003-530699P	20031216 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, P.O. BOX 1022, MINNEAPOLIS, MN, 55440-1022, US
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 1593
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 23 USPATFULL on STN
TI Immunogenic compositions and methods of use thereof
AB The present invention provides an immunogenic composition comprising lethally irradiated bacteria formulated for mucosal delivery. The present invention further provides methods of preparing a subject immunogenic composition. The present invention further provides a method of inducing an immune response in an individual to an antigen, the method generally involving administering a subject immunogenic composition to a mucosal tissue of the individual.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:202212 USPATFULL
TITLE: Immunogenic compositions and methods of use thereof
INVENTOR(S): Raz, Eyal, Del Mar, CA, UNITED STATES
Fierer, Joshua, LaJolla, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005175630	A1	20050811
APPLICATION INFO.:	US 2004-21821	A1	20041222 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-564913P	20040422 (60)
	US 2003-532786P	20031223 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BOZICEVIC, FIELD & FRANCIS LLP, 1900 UNIVERSITY AVENUE, SUITE 200, EAST PALO ALTO, CA, 94303, US
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3646
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 23 USPATFULL on STN
TI TRIF-related adaptor molecule (TRAM) and uses thereof
AB A Toll-IL-1-resistance (TIR) domain-containing adaptor-inducing IFN-β (TRIF)-related adaptor molecule (TRAM) has been identified. TRAM acts specifically in the TLR4 signaling pathway. The invention includes compounds useful for modulating TLR signaling by modulating the effects of TRAM.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:183412 USPATFULL
TITLE: TRIF-related adaptor molecule (TRAM) and uses thereof
INVENTOR(S): Fitzgerald, Katherine A., Cambridge, MA, UNITED STATES

Rowe, Daniel C., Walpole, MA, UNITED STATES
Golenbock, Douglas T., Wellesley, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005158799	A1	20050721
APPLICATION INFO.:	US 2004-968598	A1	20041018 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-512364P	20031017 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 3447
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 23 USPATFULL on STN
TI Small molecule **toll-like receptor** (TLR) antagonists
AB The invention provides methods and compositions useful for modulating signaling through Toll-like receptors. The methods involve contacting a TLR-expressing cell with a small molecule having a core structure including at least two rings. Certain of the compounds are 4-primary amino quinolines. Many of the compounds and methods are useful specifically for inhibiting immune stimulation involving at least one of TLR9, **TLR8**, TLR7, and TLR3. The methods may have use in the treatment of autoimmunity, inflammation, allergy, asthma, graft rejection, graft versus host disease, infection, sepsis, cancer, and immunodeficiency.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:138623 USPATFULL
TITLE: Small molecule **toll-like receptor** (TLR) antagonists
INVENTOR(S): Lipford, Grayson B., Watertown, MA, UNITED STATES
Forsbach, Alexandra, Ratingen, GERMANY, FEDERAL REPUBLIC OF
Zepp, Charles, Hardwick, MA, UNITED STATES
PATENT ASSIGNEE(S): Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (U.S. corporation)
Coley Pharmaceutical Group, Inc., Wellesley, MA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005119273	A1	20050602
APPLICATION INFO.:	US 2004-872196	A1	20040618 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-480588P	20030620 (60)
	US 2004-556007P	20040323 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Alan W. Steele, M.D., Ph.D., Wolf, Greenfield & Sacks, P.C., 600 Atlantic Avenue, Boston, MA, 02210, US
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1-30

NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 4382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 8 OF 23 USPATFULL on STN
TI Cell-free methods for identifying compounds that affect **toll-like receptor** 9 (TLR9) signaling
AB The invention is directed to methods for screening for a compound that affects interaction between a **Toll-like receptor** (TLR) and a ligand for the TLR. The methods involve direct measurement of interaction using, for example, surface plasmon resonance (SPR), particularly under conditions of pH that mimic those of the TLR in vivo. Compounds identified using the methods of the invention may be useful in the development of agents useful in the treatment of conditions characterized by undesirable immune activation, e.g., autoimmunity, inflammation, allergy, asthma, and transplantation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:117716 USPATFULL
TITLE: Cell-free methods for identifying compounds that affect **toll-like receptor** 9 (TLR9) signaling
INVENTOR(S): Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
Lipford, Grayson, Watertown, MA, UNITED STATES
Wagner, Hermann, Eching, GERMANY, FEDERAL REPUBLIC OF
Rutz, Mark, Muenchen, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): Coley Pharmaceutical GmbH, Langenfeld, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)
Coley Pharmaceutical Group, Inc., Wellesley, MA, UNITED STATES (non-U.S. corporation)
Technische Universitat Munchen, Muenchen, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005100983	A1	20050512
APPLICATION INFO.:	US 2004-982193	A1	20041105 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-517804P	20031106 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 835
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 9 OF 23 USPATFULL on STN
TI Methods and compositions for enhancing immune response
AB Methods and compositions for enhancing the immune response to an IRM compound by depositing within a localized tissue region an IRM depot preparation that provides an extended residence time of active IRM within the localized tissue region.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:334273 USPATFULL
TITLE: Methods and compositions for enhancing immune response
INVENTOR(S): Miller, Richard L., Maplewood, MN, UNITED STATES
Tomai, Mark A., Woodbury, MN, UNITED STATES

Kedl, Ross M., Denver, CO, UNITED STATES
Zarraga, Isidro Angelo Eleazar, Minneapolis, MN, UNITED STATES
Ortiz, Ronnie, Apple Valley, MN, UNITED STATES
Stoesz, James D., Inver Grove Heights, MN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004265351	A1	20041230
APPLICATION INFO.:	US 2004-821330	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-533703P	20031231 (60)
	US 2003-462140P	20030410 (60)
	US 2003-515256P	20031029 (60)
	US 2003-515604P	20031030 (60)
	US 2004-545424P	20040218 (60)
	US 2004-545542P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 959
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 10 OF 23 USPATFULL on STN
TI Delivery of immune response modifier compounds
AB The present invention provides immune response modifiers (IRMs) associated with (typically, attached to, and preferably, covalently attached to) macromolecular support materials. The IRM compounds in such IRM-support complexes retain biological activity. Such attachment of an IRM to a macromolecular support material provides for the localized biological activity of the IRM.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:326879 USPATFULL
TITLE: Delivery of immune response modifier compounds
INVENTOR(S): Wightman, Paul D., Woodbury, MN, UNITED STATES
Zarraga, Isidro Angelo E., Minneapolis, MN, UNITED STATES
Jing, Naiyong, Woodbury, MN, UNITED STATES
Liu, Jie J., Woodbury, MN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004258698	A1	20041223
APPLICATION INFO.:	US 2004-821335	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-462140P	20030410 (60)
	US 2004-545424P	20040218 (60)
	US 2003-515256P	20031029 (60)
	US 2004-545542P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 51
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 2407
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 11 OF 23 USPATFULL on STN
TI Methods of treating pulmonary fibrotic disorders
AB The present invention provides methods of treating airway remodeling, the methods generally involve administering an effective amount of a **Toll-like receptor agonist** to an individual suffering from airway remodeling. The present invention provides methods of treating pulmonary fibrosis, the methods generally involving administering an effective amount of a **Toll-like receptor agonist** to an individual in need thereof. The present invention further provides pharmaceutical compositions comprising a TLR **agonist** and a formulation suitable for delivery by inhalation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:315161 USPATFULL
TITLE: Methods of treating pulmonary fibrotic disorders
INVENTOR(S): Raz, Eyal, Del Mar, CA, UNITED STATES
Broide, David, San Diego, CA, UNITED STATES
Takabayashi, Kenji, San Diego, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004248837	A1	20041209	
APPLICATION INFO.:	US 2003-697817	A1	20031029	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-423035P	20021101	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BOZICEVIC, FIELD & FRANCIS LLP, 1900 UNIVERSITY AVE, SUITE 200, EAST PALO ALTO, CA, 94303
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 2304
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 12 OF 23 USPATFULL on STN
TI Delivery of immune response modifier compounds using metal-containing particulate support materials
AB The present invention provides immune response modifiers (IRMs) on particulate support materials that includes one or more metals, including alloys or complexes thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:260225 USPATFULL
TITLE: Delivery of immune response modifier compounds using metal-containing particulate support materials
INVENTOR(S): Wightman, Paul D., Woodbury, MN, UNITED STATES
Liu, Jie J., Woodbury, MN, UNITED STATES
Jing, Naiyong, Woodbury, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004202720	A1	20041014
APPLICATION INFO.:	US 2004-821319	A1	20040409 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-640904, filed on 14 Aug 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-462140P	20030410 (60)
	US 2004-545542P	20040218 (60)
	US 2003-515256P	20031029 (60)
	US 2004-545424P	20040218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
LINE COUNT: 1759
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 13 OF 23 USPATFULL on STN
TI Selective activation of cellular activities mediated through a common **toll-like receptor**
AB Methods of identifying compounds that selectively modulate cellular activities mediated by a common TLR are provided. Generally, the methods include providing an assay to detect modulation of a first cellular activity mediated by a TLR; providing an assay to detect modulation of a second cellular activity mediated by the TLR; performing each assay using a test compound; and identifying the test compound as a compound that selectively modulates at least one cellular activity of a plurality of activities mediated by a common TLR if the test compound modulates the first cellular activity to a different extent than it modulates the second TLR-mediated cellular activity. Compounds identified by such methods, pharmaceutical compositions including such compounds, and methods of treating a condition by administering such pharmaceutical compositions to a subject are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:247238 USPATFULL
TITLE: Selective activation of cellular activities mediated through a common **toll-like receptor**
INVENTOR(S): Fink, Jason R., Eagan, MN, UNITED STATES
Gupta, Shalley K., Woodbury, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004191833	A1	20040930
APPLICATION INFO.:	US 2004-807934	A1	20040324 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-457336P	20030325 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1

LINE COUNT: 1382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 14 OF 23 USPATFULL on STN
TI Selective modulation of TLR-mediated biological activity
AB Methods of identifying a compound that selectively modulates at least one TLR-mediated cellular activity are disclosed. Generally, the methods include identifying a compound as a compound that selectively modulates at least one TLR-mediated cellular activity if the compound modulates one TLR-mediated cellular activity to a different extent than it modulates a second TLR-mediated cellular activity. Compounds so identified and pharmaceutical compositions including such compounds are also disclosed. Methods of selectively modulating immune cells and methods of treating certain conditions are also provided. Such methods include administering to cells or a subject a compound that selectively modulates a TLR-mediated cellular activity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:221317 USPATFULL
TITLE: Selective modulation of TLR-mediated biological activity
INVENTOR(S): Fink, Jason R., Eagan, MN, UNITED STATES
Gorden, Keith B., Maplewood, MN, UNITED STATES
Gorski, Kevin S., White Bear Lake, MN, UNITED STATES
Gupta, Shalley K., Woodbury, MN, UNITED STATES
Qiu, Xiaohong, Rosemount, MN, UNITED STATES
Vasilakos, John P., Woodbury, MN, UNITED STATES
PATENT ASSIGNEE(S): 3M Innovative Properties Company (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004171086	A1	20040902	
APPLICATION INFO.:	US 2004-788731	A1	20040227	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-450484P	20030227	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: 3M INNOVATIVE PROPERTIES COMPANY, PO BOX 33427, ST. PAUL, MN, 55133-3427
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 1870
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 15 OF 23 USPATFULL on STN
TI **Toll-like receptor** 3 signaling agonists and antagonists
AB Compositions and methods are provided to identify, characterize, and optimize immunostimulatory compounds, their agonists and antagonists, working through TLR3.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:237844 USPATFULL
TITLE: **Toll-like receptor** 3 signaling agonists and antagonists
INVENTOR(S): Lipford, Grayson B., Dusseldorf, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE

PATENT INFORMATION: US 2003166001 A1 20030904
APPLICATION INFO.: US 2002-265072 A1 20021005 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-327520P	20011005 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3285
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 16 OF 23 USPATFULL on STN
TI Methods and products for enhancing immune responses using imidazoquinoline compounds
AB The invention involves administration of an imidazoquinoline agent in combination with another therapeutic agent. The combination of drugs may be administered in synergistic amounts or in various dosages or at various time schedules. The invention also relates to kits and compositions concerning the combination of drugs. The combinations can be used to enhance ADCC, stimulate immune responses and/or patient and treat certain disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:201378 USPATFULL
TITLE: Methods and products for enhancing immune responses using imidazoquinoline compounds
INVENTOR(S): Krieg, Arthur M., Wellesley, MA, UNITED STATES
Schetter, Christian, Hilden, GERMANY, FEDERAL REPUBLIC OF
Bratzler, Robert L., Concord, MA, UNITED STATES
Vollmer, Jorg, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF
Jurk, Marion, Dusseldorf, GERMANY, FEDERAL REPUBLIC OF
Bauer, Stefan, Muenchen, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): University of Iowa Research Foundation, Iowa City, IA, 52242 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003139364	A1	20030724
APPLICATION INFO.:	US 2002-272502	A1	20021015 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-329208P	20011012 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211
NUMBER OF CLAIMS: 87
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 25 Drawing Page(s)
LINE COUNT: 7027
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 17 OF 23 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN
TI TLR7/8-mediated activation of human NK cells results in accessory cell-dependent IFN-γ production.

AB NK cells express receptors that allow them to recognize pathogens and activate effector functions such as cytotoxicity and cytokine production. Among these receptors are the recently identified TLRs that recognize conserved pathogen structures and initiate innate immune responses. We demonstrate that human NK cells express TLR3, TLR7, and **TLR8** and that these receptors are functional. TLR3 is expressed at the cell surface where it functions as a receptor for polyinosinic acid:cytidylic acid (poly(I:C)) in a lysosomal-independent manner. TLR7/8 signaling is sensitive to chloroquine inhibition, indicating a requirement for lysosomal signaling as for other cell types. Both **R848**, an **agonist** of human TLR7 and **TLR8**, and poly(I:C) activate NK cell cytotoxicity against Daudi target cells. However, IFN-γ production is differentially regulated by these TLR agonists. In contrast to poly(I:C), **R848** stimulates significant IFN-γ production by NK cells. This is accessory cell dependent and is inhibited by addition of a neutralizing anti-IL-12 Ab. Moreover, stimulation of purified monocyte populations with **R848** results in IL-12 production, and reconstitution of purified NK cells with monocytes results in increased IFN-γ production in response to **R848**. In addition, we demonstrate that while resting NK cells do not transduce signals directly in response to **R848**, they can be primed to do so by prior exposure to either IL-2 or IFN-α. Therefore, although NK cells can be directly activated by TLRs, accessory cells play an important and sometimes essential role in the activation of effector functions such as IFN-γ production and cytotoxicity.

ACCESSION NUMBER: 2005331017 EMBASE
TITLE: TLR7/8-mediated activation of human NK cells results in accessory cell-dependent IFN-γ production.
AUTHOR: Hart O.M.; Athie-Morales V.; O'Connor G.M.; Gardiner C.M.
CORPORATE SOURCE: Dr. C.M. Gardiner, Department of Biochemistry, Trinity College, Dublin 2, Ireland. clair.gardiner@tcd.ie
SOURCE: Journal of Immunology, (1 Aug 2005) Vol. 175, No. 3, pp. 1636-1642.
Refs: 51
ISSN: 0022-1767 CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050825
Last Updated on STN: 20050825

L6 ANSWER 18 OF 23 EMBASE

TI Immunization with HIV-1 gag protein conjugated to a TLR7/8 **agonist** results in the generation of HIV-1 gag-specific Th1 and CD8(+) T cell responses.

AB One strategy to induce optimal cellular and humoral immune responses following immunization is to use vaccines or adjuvants that target dendritic cells and B cells. Activation of both cell types can be achieved using specific TLR ligands or agonists directed against their cognate receptor. In this study, we compared the ability of the TLR7/8 **agonist** R-848, which signals only via TLR7 in mice, with CpG oligodeoxynucleotides for their capacity to induce HIV-1 Gag-specific T cell and Ab responses when used as vaccine adjuvants with HIV-1 Gag protein in mice. Injection of R-848 and CpG oligodeoxynucleotides alone enhanced the innate immune responses in vivo as demonstrated by high serum levels of inflammatory cytokines, including IL-12p70 and IFN-α, and increased expression of CD80, CD86, and CD40 on CD11c(+) dendritic cells. By contrast, R-848 was a relatively poor adjuvant for inducing primary Th1 or CD8(+) T cell responses when administered with HIV-1 Gag protein.

However, when a TLR7/8 **agonist** structurally and functionally similar to R-848 was conjugated to HIV-1 Gag protein both Th1 and CD8(+) T cells responses were elicited as determined by intracellular cytokine and tetramer staining. Moreover, within the population of HIV-1 Gag-specific CD8(+) CD62(low) cells, .apprx.50% of cells expressed CD127, a marker shown to correlate with the capacity to develop into long-term memory cells. Overall, these data provide evidence that TLR7/8 agonists can be effective vaccine adjuvants for eliciting strong primary immune responses with a viral protein in vivo, provided vaccine delivery is optimized.

ACCESSION NUMBER: 2005261964 EMBASE
TITLE: Immunization with HIV-1 gag protein conjugated to a TLR7/8 **agonist** results in the generation of HIV-1 gag-specific Th1 and CD8(+) T cell responses.
AUTHOR: Wille-Reece U.; Wu C.-Y.; Flynn B.J.; Kedl R.M.; Seder R.A.
CORPORATE SOURCE: Dr. R.A. Seder, Cellular Immunology Section, Vaccine Research Center, National Institute of Allergy and Infectious Diseases, 40 Convent Drive, Bethesda, MD 20892, United States. rseder@mail.nih.gov
SOURCE: Journal of Immunology, (15 Jun 2005) Vol. 174, No. 12, pp. 7676-7683.
Refs: 44
ISSN: 0022-1767 CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050707
Last Updated on STN: 20050707

L6 ANSWER 19 OF 23 EMBASE
TI Identification and characterization of a functional, alternatively spliced **Toll-like receptor** 7 (TLR7) and genomic disruption of **TLR8** in chickens.
AB Based upon the recognition of antiviral compounds and single stranded viral RNA the Toll-like receptors TLR7 and **TLR8** are suggested to play a significant role in initiating antiviral immune responses. Here we report the molecular characterization of the chicken TLR7/8 loci which revealed an intact TLR7 gene and fragments of a **TLR8**-like gene with a 6-kilobase insertion containing chicken repeat 1 (CR1) retroviral-like insertion elements. The chicken TLR7 gene encodes a 1047-amino-acid protein with 62% identity to human TLR7 and a conserved pattern of predicted leucine-rich repeats. Highest levels of chicken TLR7 mRNA were detected in immune-related tissues and cells, especially the spleen, caecal, tonsil and splenic B cells. Alternative spliced forms of TLR7 mRNA were identified in chicken, mouse and human and expressed in similar tissues and cell types to the major form of chicken TLR7. The chicken TLR7 (+) HD11 cell line and fresh splenocytes produced elevated levels of interleukin-1β (IL-1β) mRNA after exposure to the agonists **R848** and loxoribine. Interestingly, none of the TLR7 agonists stimulated increased type I interferon (IFN) mRNA whereas poly(I:C) (a TLR3 **agonist**) up-regulated both chicken IFN-α and chicken IFN-β mRNA. In contrast, TLR7 agonists, particularly **R848** and poly(U) stimulated up-regulation of chicken IL-1β and chicken IL-8 mRNAs more effectively than poly(I:C). Stimulation of chicken TLR7 with **R848** was chloroquine sensitive, suggesting signalling within an endosomal compartment, as for mammalian TLR7. The

deletion of **TLR8** in galliforms, accompanied with the differential response after exposure to TLR7 agonists, offers insight into the evolution of vertebrate TLR function. .COPYRGT. 2005 Blackwell Publishing Ltd.

ACCESSION NUMBER: 2005159932 EMBASE
TITLE: Identification and characterization of a functional, alternatively spliced **Toll-like receptor** 7 (TLR7) and genomic disruption of **TLR8** in chickens.
AUTHOR: Philbin V.J.; Iqbal M.; Boyd Y.; Goodchild M.J.; Beal R.K.; Bumstead N.; Young J.; Smith A.L.
CORPORATE SOURCE: Dr. A.L. Smith, Division of Immunology and Pathology, Institute for Animal Health, Compton Laboratory, Newbury, Berkshire, RG20 7NN, United Kingdom. adrian.smith@bbsrc.ac.uk
SOURCE: Immunology, (2005) Vol. 114, No. 4, pp. 507-521. Refs: 66 ISSN: 0019-2805 CODEN: IMMUAM
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050505
Last Updated on STN: 20050505

L6 ANSWER 20 OF 23 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN
TI Therapeutic targeting of Toll-like receptors.
AB Toll-like receptors (TLRs) play a crucial role in innate immune response in mammals. Individual TLRs recognize microbial components that are conserved among pathogens and activate their signaling pathways. Each TLR has its own cascade of signaling pathway for exhibiting its specific responses through selective utilization of TIR domain-containing adaptors. Increasing evidence for roles of TLRs in various diseases provides us new insights for a basis of new therapies. In this review, we discuss the possibilities of therapeutics targeting TLRs in various diseases and explain potential problems associated with such approaches. .COPYRGT. 2004 Elsevier Ltd. All rights reserved.

ACCESSION NUMBER: 2005065702 EMBASE
TITLE: Therapeutic targeting of Toll-like receptors.
AUTHOR: Uematsu S.; Ishii K.J.; Akira S.
CORPORATE SOURCE: S. Akira, Department of Host Defense, Res. Inst. for Microbial Diseases, Osaka University, 3-1 Yamada-oka, Suita, Osaka 565-0871, Japan. sakira@biken.osaka-u.ac.jp
SOURCE: Drug Discovery Today: Therapeutic Strategies, (2004) Vol. 1, No. 3, pp. 299-304. Refs: 22 ISSN: 1740-6773
PUBLISHER IDENT.: S 1740-6773(04)00061-0
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050224
Last Updated on STN: 20050224

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(1) a composition (C1) comprising (IA) and an immunostimulatory nucleic acid;

(2) a composition (C2) comprising an (IA) and an antibody;

(3) a composition (C3) comprising an (IA) and a disorder-specific medicament; and

(4) screening (M4) for comparing **Toll-like receptor** (TLR) signaling activity of a test compound with TLR signaling activity of IA involves contacting a functional TLR chosen from TLR7 and **TLR8** with a reference (IA) and detecting a reference response mediated by a TLR signal transduction pathway, contacting the functional TLR with a test compound and detecting a test response mediated by a TLR signal transduction pathway and comparing the test response with reference response to compare the TLR signaling activity of the test compound with (IA).

ACTIVITY - Antiasthmatic; Cytostatic; Antimicrobial; Dermatological; Virucide.

MECHANISM OF ACTION - Stimulator of ADCC; Modulator of immune response; Inducer of antigen-specific immune response (claimed); Inducer of expression of cytokines including interferons; Stimulator of Th1 immune response; Inhibitor of production of Th2 cytokines such as IL-4, IL-5 and IL-13; Increase NK cell lytic activity; Stimulator of B cell proliferation and differentiation.

The activity of R-848 to induce IFN- alpha in monocyte-derived dendritic cells (mDCs) was evaluated as follows: unfractionated human peripheral blood mononuclear cell (PBMC), containing mDCs and plasmacytoid dendritic cells (pDCs), were incubated for 48 hours in the presence of varying concentrations of R-848 (0.01-1.0 micro g/ml), varying concentrations of CpG ODN 2006 (0.2-3.0 mu g/ml), varying concentrations of negative control ODN 5177 (5' TCCGCCCTGTGACATGCATT 3'). Staphylococcal enterotoxin B or media alone, and then the concentration of IFN- alpha in the supernatant was measured by ELISA. R-848 induced higher amounts of IFN- alpha upon incubation of human PBMC than type B CpG ODN 2006.

USE - (M1) is useful for stimulating ADCC in a subject. The antibody is chosen from anti-cancer antibody, anti-viral antibody, anti-bacterial antibody, anti-fungal antibody, anti-allergen antibody and anti-self antigen antibody. The subject has or is at risk of having a disorder chosen from asthma/allergy, infectious disease, cancer and warts. (IA) which is imidazoquinoline amine is administered prior to the antibody. (IA) is chosen from imiquimod/R-837 and S-28463/R-848. (M2) is useful for modulating an immune response in a subject who is an immunocompromised subject or elderly or an infant. The immune response is a Th1 immune response, ADCC, innate immune response, local immune response, mucosal immune response or systemic immune response. The agent is administered prior to the immunostimulatory nucleic acid. The amount is effective to modulate the immune response is a synergistic amount. The method further involves administering disorder-specific medicament chosen from cancer medicament, and asthma/allergy medicament, an infectious disease medicament and a wart medicament to the subject. The cancer medicament is chosen from chemotherapeutic agent, an immunotherapeutic agent and a cancer vaccine. The asthma/allergy medicament is chosen from steroids, immunomodulators, anti-inflammatory agents, bronchodilators, leukotriene modifiers, beta 2 agonists,and anticholinergics. The anti-microbial medicament is chosen from an anti-bacterial agent, an anti-viral agent, an anti-fungal agent, and an anti-parasitic agent. The method further involves exposing the subject to an antigen chosen from tumor antigen, viral antigen, bacterial antigen, parasitic antigen, and fungal antigen and where the immune response is an antigen-specific immune response. The subject has or is at the risk of developing an infectious disease, developing a cancer, or developing asthma/allergy. (M3) is useful for inducing an antigen-specific immune response in a subject (all claimed). (M2) is useful for treating a subject having asthma/allergy, infectious disease, cancer and warts.

ADVANTAGE - IA used in conjunction with other agents such as

antibodies, immunostimulatory nucleic acid, antigens, C8-substituted guanosines and disorder-specific medicaments provides improved results.

DESCRIPTION OF DRAWING(S) - The figure shows a bar graph depicting hTLR9-mediated activation of NF-kappa B by CpG ODN 2006, but not by R-848.

Dwg.1/20

ACCESSION NUMBER: 2003-829705 [77] WPIDS
DOC. NO. NON-CPI: N2003-662840
DOC. NO. CPI: C2003-233743
TITLE: Stimulating antibody dependent cellular cytotoxicity, modulating immune response and inducing antigen-specific immune response in subject by administering imidazoquinoline agents in conjunction with other agents.
DERWENT CLASS: B04 B05 D16 S03
INVENTOR(S): BAUER, S; BRATZLER, R L; JURK, M; KRIEG, A M; SCHETTER, C; VOLLMER, J
PATENT ASSIGNEE(S): (IOWA) UNIV IOWA RES FOUND; (COLE-N) COLEY PHARM GMBH; (COLE-N) COLEY PHARM GROUP INC
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2003139364	A1	20030724	(200377)*		112
WO 2003094836	A2	20031120	(200403)	EN	
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2002360278	A1	20031111	(200442)		
EP 1478371	A2	20041124	(200477)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
JP 2005519990	W	20050707	(200545)		158

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2003139364	A1 Provisional	US 2001-329208P	20011012
		US 2002-272502	20021015
WO 2003094836	A2	WO 2002-US33051	20021015
AU 2002360278	A1	AU 2002-360278	20021015
EP 1478371	A2	EP 2002-795524	20021015
		WO 2002-US33051	20021015
JP 2005519990	W	WO 2002-US33051	20021015
		JP 2004-502925	20021015

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002360278	A1 Based on	WO 2003094836
EP 1478371	A2 Based on	WO 2003094836
JP 2005519990	W Based on	WO 2003094836

PRIORITY APPLN. INFO: US 2001-329208P 20011012; US 2002-272502 20021015

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TI Human Immunodeficiency Virus Type 1 Activates Plasmacytoid Dendritic Cells and Concomitantly Induces the Bystander Maturation of Myeloid Dendritic Cells.

AB In this study, we analyzed the phenotypic and physiological consequences of the interaction of plasmacytoid dendritic cells (pDCs) with human immunodeficiency virus type 1 (HIV-1). pDCs are one cellular target of HIV-1 and respond to the virus by producing alpha/beta interferon (IFN-α/β) and chemokines. The outcome of this interaction, notably on the function of bystander myeloid DC (CD11c(+) DCs), remains unclear. We therefore evaluated the effects of HIV-1 exposure on these two DC subsets under various conditions. Blood-purified pDCs and CD11c(+) DCs were exposed in vitro to HIV-1, after which maturation markers, cytokine production, migratory capacity, and CD4 T-cell stimulatory capacity were analyzed. pDCs exposed to different strains of infectious or even chemically inactivated, nonreplicating HIV-1 strongly upregulated the expression of maturation markers, such as CD83 and functional CCR7, analogous to exposure to R-848, a synthetic **agonist** of **toll-like receptor**-7 and -8. In addition, HIV-1-activated pDCs produced cytokines (IFN-α and tumor necrosis factor alpha), migrated in response to CCL19 and, in coculture, matured CD11c(+) DCs, which are not directly activated by HIV. pDCs also acquired the ability to stimulate naive CD4(+) T cells, albeit less efficiently than CD11c (+) DCs. This HIV-1-induced maturation of both DC subsets may explain their disappearance from the blood of patients with high viral loads and may have important consequences on HIV-1 cellular transmission and HIV-1-specific T-cell responses.

ACCESSION NUMBER: 2004198414 EMBASE

TITLE: Human Immunodeficiency Virus Type 1 Activates Plasmacytoid Dendritic Cells and Concomitantly Induces the Bystander Maturation of Myeloid Dendritic Cells.

AUTHOR: Fonteneau J.-F.; Larsson M.; Beignon A.-S.; McKenna K.; Dasilva I.; Amara A.; Liu Y.-J.; Lifson J.D.; Littman D.R.; Bhardwaj N.

CORPORATE SOURCE: N. Bhardwaj, NYU School of Medicine, Department of Pathology, MSB507, 550 First Ave., New York, NY 10016, France. bhardn02@med.nyu.edu

SOURCE: Journal of Virology, (2004) Vol. 78, No. 10, pp. 5223-5232. Refs: 51
ISSN: 0022-538X CODEN: JOVIAM

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20040520
Last Updated on STN: 20040520

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TI Randomized, Single-Blind, Placebo-Controlled Study of Topical Application of the Immune Response Modulator **Resiquimod** in Healthy Adults.

AB **Resiquimod** is a **Toll-like receptor** 7 (TLR7) and **TLR8 agonist** that is a potent inducer of alpha interferon (IFN-α) and other cytokines. The effects of multiple applications of **resiquimod** gel were assessed in a randomized, single-blind, dose-ranging, placebo-controlled study with 41 healthy subjects. Over a 3-week period, 1-g doses of **resiquimod** or vehicle gel (3:1 randomization) were applied to a 50-cm(2) area of the upper arm according to the following regimens: 0.25% applied for 8 h two times per week, 0.05% applied for 8 h two times per week, 0.05% applied

for 8 h three times per week, and 0.01% applied for 24 h three times per week. Skin biopsy specimens were obtained prior to the application of the first dose and after the completion of application of the last dose. Dosing with 0.01 and 0.05% **resiquimod** was well tolerated, but that with 0.25% was not; a dose-response relationship for local adverse effects was observed. The level of systemic exposure during multiple topical dosings was <1% of the applied dose. A significant increase in responders after completion of application of the last dose was observed for serum IFN and the interleukin-1 (IL-1) receptor antagonist ($P < 0.01$, Fisher's exact test). Increased levels of mRNA for IL-6, IL-8, IFN-α, and Mx (an IFN-α-inducible protein) were seen in posttreatment biopsy specimens from the group receiving 0.25% **resiquimod** compared to the levels seen in specimens from the group receiving vehicle only ($P < 0.01$, Wilcoxon rank sum test). A dose-response-related increase in CD3-positive cells consistent with T-lymphocyte infiltration and a decrease in CD1a-positive cells, consistent with emigration of Langerhans' cells, were observed in treated skin. This study suggests that **resiquimod** is a potent topically active immune response modifier that significantly enhances the cutaneous immune response.

ACCESSION NUMBER: 2003493058 EMBASE
TITLE: Randomized, Single-Blind, Placebo-Controlled Study of Topical Application of the Immune Response Modulator **Resiquimod** in Healthy Adults.
AUTHOR: Sauder D.N.; Smith M.H.; Senta-McMillian T.; Soria I.; Meng T.-C.
CORPORATE SOURCE: T.-C. Meng, 3M Pharmaceuticals, 3M Center, Saint Paul, MN 55144-1000, Canada. tmeng1@mmm.com
SOURCE: Antimicrobial Agents and Chemotherapy, (2003) Vol. 47, No. 12, pp. 3846-3852.
Refs: 21
ISSN: 0066-4804 CODEN: AMACCQ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040116
Last Updated on STN: 20040116

L6 ANSWER 23 OF 23 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Stimulating antibody dependent cellular cytotoxicity, modulating immune response and inducing antigen-specific immune response in subject by administering imidazoquinoline agents in conjunction with other agents.
AN 2003-829705 [77] WPIDS
AB US2003139364 A UPAB: 20031128
NOVELTY - Stimulating (M1) antibody dependent cellular cytotoxicity (ADCC), modulating (M2) immune response and inducing (M3) antigen-specific immune response in a subject by administering an antibody, immunostimulatory nucleic acid and antigen and immunostimulatory nucleic acid respectively along with imidazoquinoline agents, is new.

DETAILED DESCRIPTION - Stimulating (M1) antibody dependent cellular cytotoxicity (ADCC) in a subject by administering an antibody and an agent (I) chosen from imidazoquinoline agent (IA) and a C8-substituted guanosine, modulating (M2) immune response in a subject by administering immunostimulatory nucleic acid and (I) and inducing (M3) antigen-specific immune response in a subject by administering an antigen, an (IA) and immunostimulatory nucleic acid.

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All: 133 Review: 4

Items 1 - 20 of 133 Page 1 of 7 Next

1: Wang Y, Abel K, Lantz K, Krieg AM, McChesney MB, Miller CJ. Related Articles, Links

The Toll-Like Receptor 7 (TLR7) Agonist, Imiquimod, and the TLR9 Agonist, CpG ODN, Induce Antiviral Cytokines and Chemokines but Do Not Prevent Vaginal Transmission of Simian Immunodeficiency Virus When Applied Intravaginally to Rhesus Macaques.
J Virol. 2005 Nov;79(22):14355-70.
PMID: 16254370 [PubMed - in process]

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Generation and Maturation of Dendritic Cells for Clinical Application Under Serum-Free Conditions.
J Immunother. 2005 November/December;28(6):599-609.
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FASEB J. 2005 Oct 11; [Epub ahead of print]
PMID: 16219804 [PubMed - as supplied by publisher]

4: Wille-Reece U, Flynn BJ, Lore K, Koup RA, Kedl RM, Mattapallil JJ, Weiss WR, Roederer M, Seder RA. Related Articles, Links

HIV Gag protein conjugated to a Toll-like receptor 7/8 agonist improves the magnitude and quality of Th1 and CD8+ T cell responses in nonhuman primates.
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Evidence for dissociation of TLR mRNA expression and TLR agonist-mediated functions in bovine macrophages.
Vet Immunol Immunopathol. 2005 Oct 6; [Epub ahead of print]
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Modulation of atherosclerosis in mice by Toll-like receptor 2.
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Related Resources
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Consumer Health
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ClinicalTrials.gov
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DOCUMENT-IDENTIFIER: US 20040228847 A1

TITLE: Progenitor cells and methods of using same

PUBLICATION-DATE: November 18, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Goldschmidt-Clermont, Pascal J.	Chapel Hill	NC	US
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US-CL-CURRENT: 424/93.21; 424/93.71

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PGPUB-DOCUMENT-NUMBER: 20030022302
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TITLE: Toll-like receptor

PUBLICATION-DATE: January 30, 2003

INVENTOR-INFORMATION:

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US-CL-CURRENT: 435/69.1; 435/320.1, 435/325, 530/350, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040023870 A1

TITLE: Methods of therapy and diagnosis using targeting of cells that express toll-like receptor proteins

PUBLICATION-DATE: February 5, 2004

INVENTOR-INFORMATION:

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US-CL-CURRENT: 514/12; 424/144.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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